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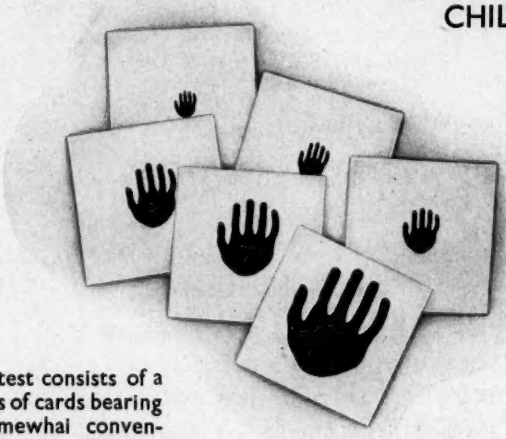
SJÖGREN'S VISUAL ACUITY TEST CARDS FOR CHILDREN



This test consists of a series of cards bearing a somewhai conventionalized hand drawn so that the fingers and the intervening spaces represent the usual Snellen angle of one minute of arc at six metres.

The method of procedure advocated by Dr. Sjögren is for the examiner to hold up a card at a distance of six metres from the patient, the patient then holds up his hand with the fingers pointing in the same direction as on the card. When the smallest figure for which the correct position of the fingers can be indicated has been found Snellen figure on that card will show the resultant visual acuity. This should be checked by turning the card to show the fingers pointing in different directions. When examining small children the procedure should be demonstrated to them a close range with a larger figure. Experience has proved that children down to the age of two years can easily understand the test.

Brit. J. Ophthal. (1954), **38**, 513.

COMMUNICATIONS

VASCULAR ANATOMY OF THE CHOROID IN RELATION TO SELECTIVE LOCALIZATION OF OCULAR DISEASE*

BY

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THE choroidal coat of the eye consists of a dense continuous network of vessels and yet it is a striking feature of choroidal disease that the lesions often remain remarkably circumscribed. It is the purpose of this paper to review the problem of selective localization of pathological changes within the choroid in relation to its vascular anatomy, with particular reference to certain findings (Wybar, 1954) recently made during a study of Neoprene latex casts of the choroidal vessels of the human eye prepared by the intravascular injection technique of Ashton (1952). The cohesive and elastic properties of Neoprene permit dissection of individual vessels or capillary areas from the main vascular mass, and these can then be studied in detail using a wide-field stereoscopic microscope with direct illumination from a high-power low-voltage filament lamp. This technique represents a distinct advance on former dye injection methods, particularly in such a densely vascular tissue as the choroid.

Previous Theories in the Literature

Theory of Division into Sectors

The experimental investigations of Wagenmann (1890) on the rabbit gave rise to the belief that the short posterior ciliary arteries are functional end-arteries and that the vessels within the choroid have a strictly segmental distribution. Wagenmann found that, when he sectioned a few short posterior ciliary arteries behind the globe in the living animal, a localized area of choroidal atrophy occurred, which corresponded in situation to the field of distribution of the affected vessels and was sharply demarcated from the surrounding healthy choroid. There was a tendency for some of the vessels in the affected zone to re-open within a few days, but in many of the animals a small sector of permanent vascular obliteration remained. The detailed study of the choroidal circulation by Leber (1903) contributed to this concept by the emphasis which he laid on the absence of anastomoses between the various branches of the short posterior ciliary arteries except in the immediate vicinity of the optic disc and in the extreme periphery of the choroid.

Certain clinical observations have been advanced to substantiate Wagenmann's findings of a segmental pattern of the choroidal vascular supply. A wedge-shaped area of choroidal degeneration was described by Coats (1907) in a case of posterior scleritis due, he presumed, to occlusion of a short posterior ciliary artery in the sclera when this tissue was inflamed. Hepburn

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(1912) also believed that the blockage of a short posterior ciliary artery might produce a wedge-shaped defect in the choroid, although he based this conclusion entirely on an interpretation of the changes in the visual fields in certain cases showing small deep foci of choroiditis. In such cases he found a sector-shaped scotoma extending from the focus of choroiditis to the periphery of the visual field, and he considered that this indicated a circulatory failure within the whole underlying sector of choroid due to the involvement of a short posterior ciliary artery in the inflammatory focus.

Theory of Division into Zones

In addition to the concept that the choroid is divided into discrete radially arranged sectors, each of which extends from the peripapillary region to the peripheral limit of the choroid, it has been postulated that there are several specialized and distinct zones within the choroid, which include the equator, the periphery, the region underlying the macula, and the peripapillary zone. The anatomical and clinical factors which have led to this view are reviewed below.

Equatorial Choroid.—This zone was considered by Leber (1903) to be a peculiar region of the choroid in that it represented the meeting place of the terminal twigs of the short posterior ciliary arteries running forwards, and of the recurrent choroidal arteries from the anterior part of the uveal tract running backwards; Leber regarded these two systems as fairly separate entities, despite their association at the equator through an intervening capillary anastomosis. This view of an ineffective anastomosis at the equator was also held by Gonin (1903) and Nettleship (1903), who considered that the resulting poverty in blood supply in that region rendered it particularly vulnerable to pathological conditions affecting the choroidal vascular bed. This conclusion was derived from a study of the changes in the visual fields in cases of retinitis pigmentosa, and was based on the hypothesis that the retinal degeneration resulted from a defect in the underlying choroidal circulation. In this way the ring scotoma, the characteristically early visual field change in retinitis pigmentosa, was interpreted as indicating a preferential disturbance of the equatorial part of the choroid.

Peripheral Choroid.—The zone in the extreme periphery of the fundus has been considered to be a specialized area; indeed, this is the logical outcome of an acceptance of the view that the anterior vascular supply is partially isolated from the rest of the choroid by the imperfect anastomosis between the two vascular systems at the equator. Evidence suggesting a peculiarity in the structure of this region of the choroid was advanced by Hancock (1908), who pointed out that the function of the extreme periphery of the retina may be retained in certain cases of total occlusion of the central retinal artery. He regarded this as an indication of a specialization of the chorio-capillaris in the extreme periphery, so that it is capable of nourishing the whole thickness of the retina, in contrast to the chorio-capillaris elsewhere, which can maintain the viability of the outer retinal layers only. The fact that transudate

can filter through the whole thickness of the peripheral part of the retina has led to the suggestion that the anterior part of the choroid may contribute to the formation of aqueous humour (Duke-Elder, 1948); and this may be a further indication of a peculiarity of that part of the uvea.

Sub-Macular Choroid.—This area has been considered to be peculiar in two respects: first, in that its capillaries are more densely packed with a corresponding reduction in the inter-capillary spaces (Leber, 1903; Lauber, 1931), and second, in that it is more vascular because of a greater aggregation of vessels within its area (Wolfrum, 1908; Salzmann, 1912; Lauber, 1936; Mörike, 1949). The concept of a specialized structure of the choroidal vascular bed at the macula is of particular interest in view of the frequency with which certain pathological processes affecting the uveal tract remain confined to the posterior pole of the eye. For example, the choroidal haemorrhage which impairs the function of the retina in certain senile eyes and in advanced myopia occurs characteristically in this region, and cystoid degenerative changes of the retina, which follow degeneration of the underlying chorio-capillaris, is characteristically limited to the macula; indeed it has been shown pathologically (Ashton, 1953) that extensive atrophy of the chorio-capillaris in the macular and paramacular regions may occur in central senile areolar choroidal sclerosis without any similar involvement of the rest of the choroid. The localization of this disease to the central part of the fundus prompted Nettleship (1903) to put forward the hypothesis that it is based on a narrowing of the lumen of the short posterior ciliary arteries supplying the choroid at the posterior pole.

Peripapillary Choroid.—The area surrounding the optic disc has also been considered to be a specialized zone (Duke-Elder, 1940). A continuous arterial network in this region, composed of arterioles which do not participate in the vascular supply of the rest of the choroid, was described by Marín-Amat (1952), who suggested that certain forms of contusion of the eye may rupture these fine vessels by suddenly stretching and twisting the globe. Involvement of this vascular ring would thus be followed by the development of a peripapillary choroidal atrophy (Bartolozzi, 1952).

Objections to these Theories

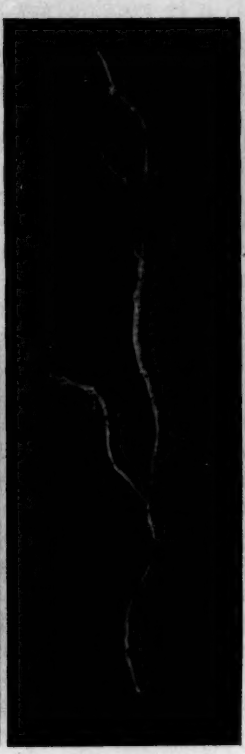
All this evidence, apparently pointing to a rigid segmentation of the posterior ciliary vessels and to a regional characterization of certain areas of the choroid and its capillary network, forms a particularly attractive thesis in considering the selective localization of choroidal disease, but there are findings which are opposed to such a concept:

(1) *There is doubt regarding the validity of the hypothesis that the arteries in the choroid maintain a rigid independence of one another, and that they, therefore, have the characteristics of end-arteries.*

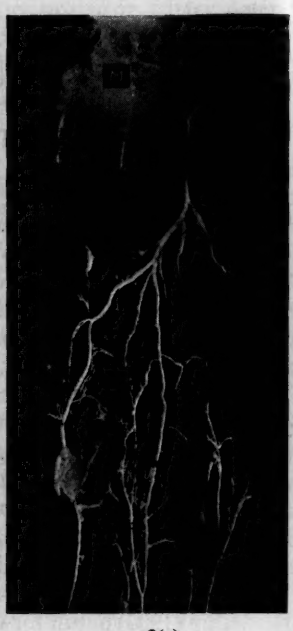
Each short posterior ciliary artery, as it perforates the sclera in the posterior part of the globe, breaks up into many branches which run forward in narrow

FIG. 1.—Neoprene casts of two short posterior ciliary arterial branches showing localized distribution of their terminal arteriolar-capillary network, (*a*) in more posterior part of choroid, (*b*) in more anterior part reaching forward to the equator. (Dissected specimens). ×12.

1(*a*)

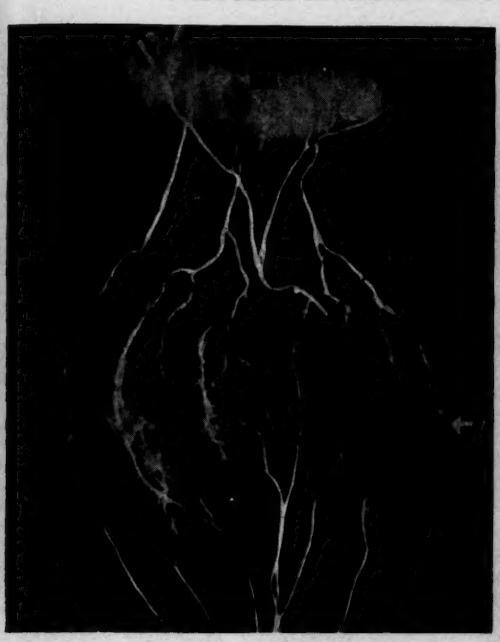


1(*b*)



2(*a*)

sectors of the choroid and terminate in arteriolar-capillary networks at particular points between the peripapillary region and the equator of the eye (Figs 1*a* and 1*b*). These arteriolar-capillary networks, however, do not form isolated units because they are linked with each other through the chorio-capillaris, which lies as a continuous capillary layer on the inner surface of the choroid. Furthermore, many of the short posterior ciliary arteries which run forward to the equator come into contact there with the recurrent choroidal arteries arising from three sources: the major arterial circle of the iris (Fig. 2*a*), the long posterior ciliary arteries (Fig. 2*b*, opposite), and the anterior ciliary arteries (Fig. 2*c*, opposite); and, although this anastomosis may be of a capillary nature (Fig. 3, opposite), it has been shown (Wybar, 1954) that it is not uncommon for the opposing vessels to be directly continuous with one another (Figs 4*a* and 4*b*, overleaf). The anterior part of the uveal circulation and the close anastomosis between the posterior and recurrent choroidal circulations at the equator, therefore, provide another pathway through which individual arteriolar-capillary networks may be linked to one another.



2(*b*)

2(*c*)

FIG. 2.—Neoprene casts of three strips of uvea showing recurrent choroidal arteries (R) arising:
(*a*) from the major arterial circle of the iris (M);
(*b*) from a long posterior ciliary artery (L);
(*c*) from an anterior ciliary artery (A).
The terminal parts of several short posterior ciliary arterial branches (S) are seen below. Note direct continuity of the two arterial systems at equator (E), although in places there is an intervening capillary network. (Dissected specimens). (*a*) × 8; (*b*) × 10; (*c*) × 8. (Wybar, 1954).

An attempt has been made to study this aspect of the choroidal circulation by injecting the choroid with Neoprene latex or with Indian ink through the ophthalmic artery after occlusion of a single short posterior ciliary artery in the orbit. In four eyes, two injected with Neoprene

FIG. 3.—Neoprene cast of terminal parts of recurrent choroidal artery (R), and of short posterior ciliary arterial branch (S), showing intervening capillary network at equator (E). (Dissected specimen). × 19. (Wybar, 1954).

FIG. 4(*a*).—Neoprene cast of terminal parts of recurrent choroidal arteries (R), and of short posterior ciliary arterial branches (S), showing direct continuity of two arterial systems at equator (E). (Dissected specimen). ×19. (Wybar, 1954.)

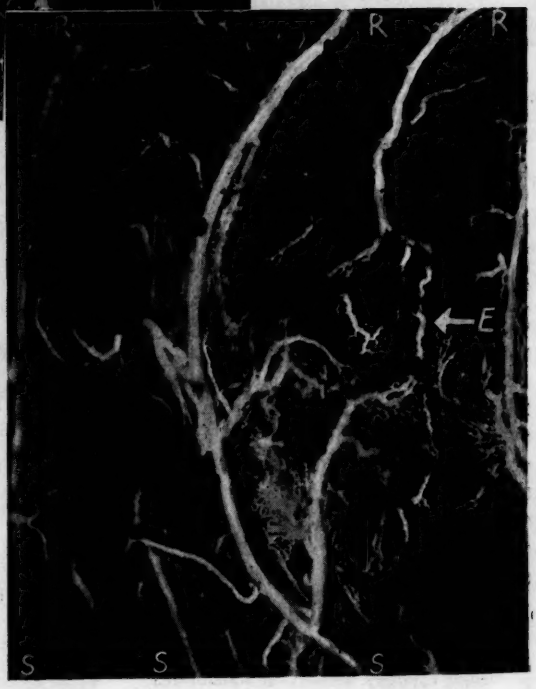


FIG. 4(*b*).—Neoprene cast of terminal parts of recurrent choroidal arteries (R), and of short posterior ciliary arterial branches (S), showing direct continuity of two arterial systems at equator (E). (Dissected specimen). ×19. Taken from left-hand side of equatorial region of Fig. 2(*b*).

FIG. 5.—Neoprene injection of part of choroid lying within globe, showing large incompletely injected sector (below) corresponding in situation to area of distribution of short posterior ciliary artery occluded before injection. ×4·5. (Anterior part of globe not available for study).

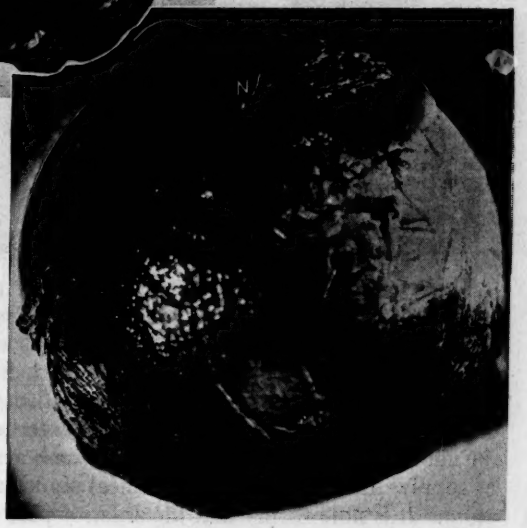


FIG. 6.—Indian ink injection of part of uveal tract, showing large incompletely injected sector corresponding in situation to area of distribution of short posterior ciliary artery occluded before injection. Position of this artery relative to globe indicated by notch in iris (N). ×4·5.

and two with Indian ink, the choroid was completely filled despite the ligation of the vessel; this would be expected, in view of the ease with which the injected material may pass from the posterior ciliary arteries through the equator to the anterior part of the uveal tract, where it is redistributed by the recurrent choroidal arteries to any sector of the choroid which has been inadequately filled from its own posterior ciliary vessels or from the neighbouring choriocapillaris. The results of this study were not consistent, however, because in three other eyes, two injected with Neoprene and one with Indian ink, a partial sectorial filling defect occurred in the choroid, corresponding in situation to the blocked short posterior ciliary artery (Figs 5 and 6).

Furthermore, in a different approach to the problem, Indian ink was injected directly into a single short posterior ciliary artery immediately behind the globe in two eyes, and although more than two-thirds of the choroid became filled with the dye, the entire choroid did not become injected in either eye (Fig. 7).

FIG. 7.—Indian ink injection of extensive area of uveal tract after injection into single short posterior ciliary artery. Position of this artery relative to globe indicated by notch in iris (N). ×3.

These inconsistencies are probably purely expressions of the degree of success of the injection technique. In the first place the injected material passes into the arteriolar-capillary networks related directly to the vessels which are being filled, but, if the injection is continued further, the zone dependent on the occluded or non-injected vessels will become filled by way of the alternative pathways through the anterior part of the eye and through the chorio-capillaris, so that, although each arterial branch has its own area of supply, there is no rigid anatomical segmentation of the choroidal circulation. It is noteworthy that the anterior part of the uveal tract was incompletely injected in the eyes which showed a sectorial filling defect (Fig. 6). It would be expected, therefore, on anatomical grounds, that in the event of a circulatory failure within a choroidal sector following occlusion of a short posterior ciliary artery, a restoration of circulation could occur from the other ciliary vessels.

This doubt regarding the rigid segmentation of the choroidal vessels was expressed by Nicholls (1938), who did not confirm the experimental findings of Wagenmann (1890). He found no structural changes within the choroid of the rabbit after occlusion of the short posterior ciliary arteries, unless at least two-thirds of the arteries were affected, in which case a phthisical condition of the eye was more likely to develop than a sectorial area of choroidal atrophy. It would also appear

that the clinical cases which have been cited to illustrate the existence of choroidal segmentation are based on hypotheses which may not be valid. In the case described by Coats (1907), it is more likely that the wedge-shaped area of choroidal atrophy, found in association with a patch of posterior scleritis, was due to involvement of the choroidal sector in the scleral inflammation with consequent obliteration of the vessels and capillaries, rather than to indirect circulatory failure in the sector from blockage of a short posterior ciliary artery within the sclera. An area of choroid will not become atrophic unless its chorio-capillaris is affected, so that the pathogenesis suggested by Coats is valid only if the thrombotic process extends into the chorio-capillaris of the affected sector.

In the case described by Hepburn (1912), the sector-shaped scotoma extending to the periphery of the visual field, following a circumscribed patch of deep choroiditis, may be explained solely on the basis of involvement of the overlying retina in the inflammatory process, without postulating any circulatory failure within the whole sector of the choroid corresponding in shape to the visual field defect. If the choroidal inflammation involves only the outer layers of the overlying retina, the defect in the visual field is a localized scotoma corresponding in size and position to the focus of choroiditis, but if the inner retinal layers are also involved, the function of a whole sector of retina is affected, with the production of a scotoma extending to the periphery of the visual field. Further evidence that this is the probable interpretation of such cases was brought forward by Wolff and Penman (1950), who demonstrated that a narrow functional zone may remain between the localized and sectorial field defects. This would not be so if the field loss were due to a circulatory failure in a complete choroidal sector, but it can be explained on the basis of an involvement of the retinal nerve fibre layer.

(2) *There is doubt regarding the validity of the hypothesis that the equator is a specialized area of the choroid in that it represents a zone of ineffective anastomoses between the posterior and recurrent choroidal circulations.*

It has been shown above that many of the vessels of the two opposing circulations are in direct continuity with one another at the equator, and that there is no evidence of a defective circulation in this region (Figs 2*a*, 2*b*, 2*c*, and 4). The concept of an ineffective anastomosis at the equator, although partly based on the anatomical studies of Leber (1903), was derived primarily from an interpretation of the visual field changes in retinitis pigmentosa, with the basic supposition that the retinal changes are secondary to a choroidal degeneration (Gonin, 1903; Nettleship, 1903). It is now known, however, that, although patches of choroidal angiosclerosis may occur in retinitis pigmentosa (Greeves, 1912), they are not an essential part of the pathological picture (Cogan, 1950), and may indeed be completely absent (Collins, 1919; Verhoeff, 1931). Experimental evidence of a hereditary pigmentary degeneration of the retina in rats (Bourne, Campbell, and Tansley, 1938) and in dogs (Parry, 1953), a condition which appears to be akin to retinitis pigmentosa, also indicates a primary retinal disturbance without any related choroidal lesions. There is, therefore, no sound basis for drawing conclusions regarding the state of the choroidal circulation from a study of the retina in retinitis pigmentosa; and the finding of an effective anastomosis at the equator does not conflict with modern views on the pathogenesis of the disease.

In this connexion it should be noted that the view put forward by Hepburn (1910), that the choroid is a series of terminal vascular systems, which, in disease, may be separately affected without necessarily involving neighbouring vessels, was also based to a certain extent on an interpretation of the visual field changes in retinitis pigmentosa in the belief that the disease is a primary choroidal affection. The choroid was divided by Hepburn into three main zones: macular, mid-peripheral, and extreme peripheral, but it should be remembered that these terms were used to denote the ophthalmoscopically visible choroid and not in any exact anatomical sense. The " macular zone " extends from the fixation point to the 20° circle concentric with the fixation point, the " mid-peripheral zone " from the 20° to the 60° circles, and the "extreme peripheral zone" from the 60° circle to the region of the anatomical equator; Hepburn regarded each zone as a separate entity with only a few intervening capillary anastomoses. His conclusions, however, are not based on sound principles as has been shown above.

(3) *There is doubt regarding the validity of the hypothesis that the periphery of the choroid is a distinct zone isolated from the rest of the choroid.*

Indeed, the idea would appear untenable, since direct anastomoses have been shown to link the posterior and recurrent choroidal circulations in man at the equator, and since after optico-ciliary resection many areas of the choroid behind the equator may be spared (Komoto, 1915; Cogan, 1950), particularly if the anterior ciliary arteries are left intact (Berlin, 1871; Studer, 1906; Birch-Hirschfeld, 1910). Nor is it necessary to endow the choroidal periphery with any peculiar functional ability in order to explain the fact that after blockage of the central retinal artery the whole thickness of the retina in the extreme periphery may remain viable, whereas elsewhere the inner part of the retina becomes degenerate. It has been shown by Michaelson (1950) that the choroid in general is able to supply nourishment to the retina to a depth of about 130 microns. This thickness covers the pigment layer, the layer of rods and cones, and the outer nuclear and outer plexiform layers over the whole of the retina, except in the extreme periphery and at the macula where the entire retina narrows to about that thickness. It is not surprising, therefore, that the retina in the extreme periphery can be nourished exclusively by the choroid, and it is understandable that part of the transudate from the anterior part of the choroid may filter through the attenuated peripheral retina to contribute to the formation of aqueous humour; but these unusual features appear to be attributable to peculiarities of retinal rather than of choroidal structure. This is in keeping with the failure to find any specific features in the peripheral part of the chorio-capillaris in an examination of casts of the choroid prepared by the intravascular injection of Neoprene. It is true that the capillaries of the choroid are not uniform in calibre throughout the chorio-capillaris; posteriorly they are smaller with a closer capillary network, and anteriorly they are wider with a more open network, but this variation in calibre comes about gradually as one passes from the central to the peripheral parts of the choroid, and there is no sharp distinction between individual areas (Fig. 8, opposite).

FIG. 8.—Neoprene cast of chorio-capillaris showing increase in calibre of capillaries and opening up of intercapillary spaces in passing from posterior to anterior parts of choroid, that is, from areas marked 1 to 4. (Dissected specimen.) ×5; insets ×24. (Wybar, 1954.)

(4) *There is doubt regarding the validity of the hypothesis that the choroid underlying the macula has distinctive features, at least as far as its capillary layer is concerned.*

An examination of casts of the choroid injected with Neoprene did not reveal any structural differences between the chorio-capillaris underlying the macula and any other part of the chorio-capillaris situated an equivalent distance from the optic disc. There is, of course, a marked difference between scattered areas of the chorio-capillaris, in view of the gradual change in the calibre of the capillaries in passing from the central to the peripheral parts of the choroid; but it would appear that the density and calibre of the capillaries are purely expressions of the distance of the capillaries from the optic disc without any reference to particular areas. Vilstrup (1952), in an examination of casts of the chorio-capillaris in the rabbit prepared by intravascular injection of gelatin and of a plastic substance, did not detect any peculiar features of the chorio-capillaris underlying the macula, and Mörike (1949) found a uniform thickness of the chorio-capillaris throughout the choroid.

The submacular part of the choroid has a certain distinction, however, owing to the greater aggregation of arterial branches from the posterior ciliary arteries which lie within its area (Fig. 9, overleaf); in Neoprene casts this feature is only apparent after removal of the extensive network of choroidal veins which sweep round in the posterior part of the globe before passing to one of the main vortex veins (Fig. 10, overleaf).

FIG. 9.—Neoprene cast of part of choroid surrounding optic disc (O), showing a greater number of arterial branches in submacular area (M). (Dissected specimen in which majority of choroidal veins have been removed). ×6.

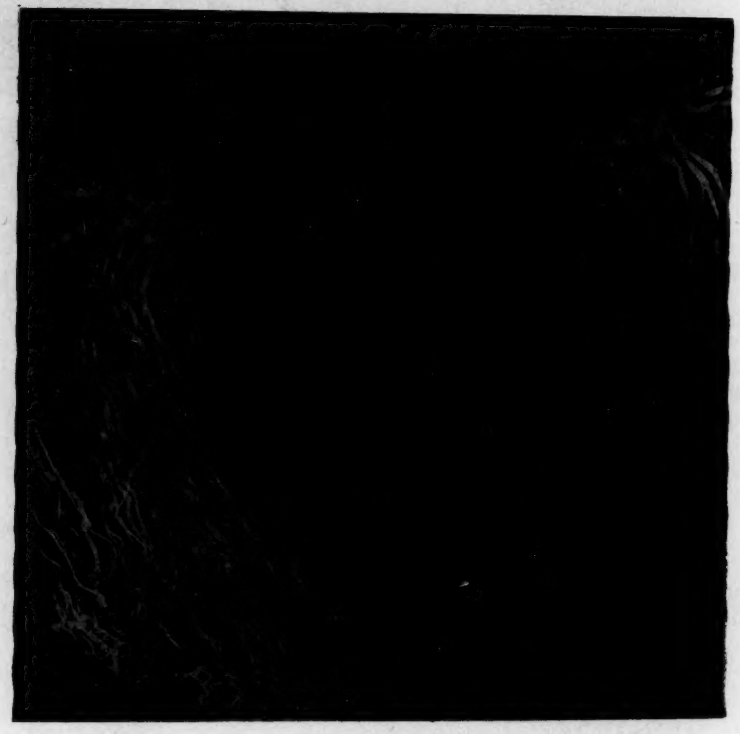


FIG. 10.—Neoprene cast of choroidal veins showing two main vortex vein systems (V.O.) and one subsidiary vortex vein system (V.S.). Note sweeping round of certain venules in peripapillary region (O) as they run to the vortex veins. (Dissected specimen.) ×6. (Wybar, 1954).

It is interesting to speculate whether the increased arterial network of the submacular choroid is due merely to the fact that a greater number of posterior ciliary arteries perforate the globe in the neighbourhood of the posterior pole, or whether it arises as a physiological response to the higher metabolic demands of the specialized macula as postulated by Nettleship (1903). The latter view is supported by Mörike (1949), who found a well-marked thickening of the choroid under each macula in the bifoveal eye of the sea-swallow, despite the remoteness of either macula from the site of perforation of the ciliary arteries. On the other hand, from an anatomical point of view as has been mentioned above, the attenuation of the macular part of the retina is such that it may be nourished exclusively by the normal choroid, and in any event it is unlikely that a mere increase in the number of arteries affects the nutritive function of an area of choroid in the absence of specialized structural changes in the associated part of the chorio-capillaris.

(5) *There is doubt regarding the validity of the hypothesis that the peripapillary region of the choroid is supplied by an exclusive system of arteries divorced from the rest of the choroid.*

It is certainly true that within this area there are to be found many fine arterial branches which arise directly from the arterial circle of Zinn, as it lies within the sclera encircling the optic nerve, but many other fine arterial branches are derived from the short posterior ciliary arteries as they perforate the globe, and these cannot be considered to form an isolated part of the choroidal circulation. Furthermore, the capillaries in the peripapillary region are directly continuous with the main mass of the chorio-capillaris, so that there is no isolation of the peripapillary choroidal capillaries.

There does not appear, therefore, to be any clear-cut anatomical evidence to support the view that the selective localization of choroidal disease is attributable either to the involvement of isolated ciliary arteries or to peculiarities in the vascular architecture of certain parts of the choroid, and yet it is an undoubted feature of choroidal disease that the lesions tend to remain circumscribed. An area of choroid ceases to function only when the integrity of its capillaries is affected, and it has been suggested by Ashton (1953) that capillary damage may be brought about primarily by an increase in the normal tissue pressure within a localized region of the choroid. A likely cause for such an increase in pressure is a hæmorrhagic or inflammatory extravasation within the choroid, which exerts a marked effect on the choroidal vascular bed owing to the limitation of expansion imposed on the choroid by the normal force of the intraocular pressure acting against the unyielding sclera. The capillaries, by virtue of their delicate structure, are more readily affected than the other choroidal vessels by such a compressive force. This hypothesis provides a possible explanation for the isolated circular patches of choroidal capillary degeneration which occur in certain inflammatory and degenerative conditions, but it does not explain why such exudative or hæmorrhagic tendencies should so often occur in particular regions.

Summary

(1) Casts of the choroidal vessels were prepared by the intravascular injection of Neoprene latex, and, owing to the cohesive and elastic properties of this substance, individual vessels and isolated capillary areas were able to be dissected from the main vascular mass and examined in detail.

(2) The short posterior ciliary arteries are segmentally arranged, and each branch supplies a localized zone of the choroid with an arteriolar-capillary network, but there is no anatomical evidence for the concept that these arteries are true end-arteries.

(3) The meeting place at the equator of the posterior choroidal circulation (short posterior ciliary arteries) and anterior choroidal circulation (recurrent arteries from the major arterial circle of the iris, from the long posterior ciliary arteries, and from the anterior ciliary arteries) may be marked by an intervening capillary network, but many of the vessels in the anastomosing circulations are in direct continuity with one another. This is contrary to the view that the equator is a zone of defective choroidal vascularization, and also to the view that the anterior part of the choroid is isolated from the rest of the structure.

(4) The size and density of the vessels in the chorio-capillaris vary in different regions according to their distance from the optic disc, but there is no evidence for the view that the chorio-capillaris underlying the macula has distinctive anatomical features.

(5) There is no anatomical evidence to suggest that the selective localization of choroidal disease is purely an expression of anatomical peculiarities of certain vascular districts.

I should like to thank Dr. Norman Ashton for introducing me to the technique of Neoprene injection and for his encouragement and advice throughout this investigation. I am indebted to Messrs. G. Knight and A. McNeil and to the Medical Illustration Department at the Institute of Ophthalmology for their help in the preparation of the casts and photographs.

REFERENCES

ASHTON, N. (1952). *British Journal of Ophthalmology*, **36**, 465.
——— (1953). *Ibid.*, **37**, 140.
BARTOLOZZI, R. (1952). *Arch. Soc. oftal. hisp.-amer.*, **12**, 692.
BERLIN, R. (1871). *Klin. Mbl. Augenheilk.*, **9**, 278.
BIRCH-HIRSCHFELD, A. (1910). *Z. Augenheilk.*, **24**, 193.
BOURNE, M. C., CAMPBELL, D. A., and TANSLEY, K. (1938). *British Journal of Ophthalmology*, **22**, 613.
COATS, G. (1907). *Trans. ophthal. Soc. U.K.*, **27**, 135.
COGAN, D. G. (1950). *Trans. Amer. Acad. Ophthal. Otolaryng.*, **54**, 629.
COLLINS, E. T. (1919). *Trans. ophthal. Soc. U.K.*, **39**, 165.
DUKE-ELDER, S. (1940). "Text-book of Ophthalmology", vol. 3, p. 2102. Kimpton, London.
——— (1948). *Trans. ophthal. Soc. U.K.*, **68**, 413.
GONIN, J. (1903). *Ann. oculist. (Paris)*, **129**, 24.
GREEVES, R. A. (1912). *Roy. Lond. ophthal. Hosp. Rep.*, **18**, 301.
HANCOCK, W. I. (1908). *Ibid.*, **17**, 428.
HEPBURN, M. L. (1910). *Ibid.*, **18**, 92.
——— (1912). *Trans. ophthal. Soc. U.K.*, **32**, 361.
KOMOTO, G. (1915). *Ibid.*, **35**, 295.

LAUBER, H. (1931). *In* "Graefe-Saemisch Handbuch der gesamten Augenheilkunde", Bd. 1, Abt. 2. Springer, Berlin.
—— (1936). *In* "Handbuch der microskopischen Anatomie des Menschen", ed. W. von Möllendorff, vol. 3, pt. 2. Springer, Berlin.
LEBER, T. (1903). *In* "Graefe-Saemisch Handbuch der gesamten Augenheilkunde", 2nd. ed., Bd. 2, Abt. 2. Springer, Berlin.
MARÍN-AMAT, M. (1952). *Amer. J. Ophthal.*, **35**, 1700.
MICHAELSON, I. C. (1950). "XVI Concilium ophthalmologicum 1950, Britannia", vol. 1, p. 605.
MÖRIKE, K. D. (1949). *Klin. Mbl. Augenheilk.*, **114**, 308.
NETTLESHIP, E. (1903). *Roy. Lond. ophthal. Hosp. Rep.*, **15**, 189.
NICHOLLS, J. V. V. (1938). *British Journal of Ophthalmology*, **22**, 672.
PARRY, H. B. (1953). *Ibid.*, **37**, 487.
SALZMANN, M. (1912). "The Anatomy and Histology of the Human Eyeball", p. 57. University of Chicago Press.
STUDER, T. F. (1906). *Arch. Ophthal.* (*Chicago*), **35**, 333.
VERHOEFF, F. H. (1931). *Ibid.*, **5**, 392.
VILSTRUP, G. (1952). "Studies on the Choroidal Circulation". Munksgaard, Copenhagen.
WAGENMANN, A. (1890). *v. Graefes Arch. Ophthal.*, **36**, pt. 4, p. 1.
WOLFF, E., and PENMAN, G. G. (1950). "XVI Concilium ophthalmologicum 1950, Britannia", vol. 1, p. 625.
WOLFRUM, M. (1908). *v. Graefes Arch. Ophthal.*, **67**, 307.
WYBAR, K. C. (1954). *J. Anat.* (*Lond.*), **88**, 94.

Brit. J. Ophthal. (1954) 38, 528.

ELECTROMYOGRAPHIC STUDY OF CONDITIONS INVOLVING LIMITED MOBILITY OF THE EYE, CHIEFLY DUE TO NEUROGENIC PARESES*

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ELECTROMYOGRAPHY has supplied us with information of great importance in the understanding of the physiology of the muscular system under normal and diseased conditions. Electromyography (EMG) has also become a routine clinical method that is widely used in diagnosis, particularly of pareses.

Not until quite recently was a method devised of obtaining electromyographic recordings from the extrinsic muscles of the human eye (Björk, 1952; Björk and Kugelberg, 1953a). Certain conditions characteristic of the ocular muscles were thereby demonstrated.

Thus the action potentials are smaller than in other human muscles (duration $1 \cdot 60 \pm 0 \cdot 06$ milliseconds in the recti). They attain far higher frequencies and recruitment is more rapid than in other muscles (Björk and Kugelberg, 1953a).

It was further shown that, when the eye is in the primary position, all the recti muscles and the levator palpebrae superioris manifest a considerable activity. In simultaneous recording from the internal and external recti it has been shown that, on outward gaze, the activity in the agonist increases greatly, while it falls successively in the antagonist but does not usually disappear even in the extreme outward position (Björk and Kugelberg, 1953b).

A preliminary account of electromyographic studies of paretic eye muscles has been published earlier (Björk, 1952). In the present paper a more detailed report will be given of investigations into cases of paresis due to a lesion of the peripheral motor neuron. Some cases have also been included in which such a lesion has been questioned without a distinct diagnosis being possible by the methods used hitherto.

Neurogenic pareses in other muscles have been studied by many authors, and the myographic picture has therefore already been thoroughly analysed. In lesions of anterior horn cells or motor nerve fibres there is a falling off in the number of action potentials which is largely proportional to the number of nerve fibres that have been put out of function and also to the diminution in muscular force (Lindsley, 1935; Seyffarth, 1938, 1940).

If the lesion is so severe that nerve fibres degenerate, fibrillations occur after 2 to 3 weeks (Denny-Brown and Pennybacker, 1938; Weddell and others, 1944). They consist of small potentials of 1–2 m.sec. duration with an

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irregular or regular fairly slow frequency, and are independent of the voluntary contraction. They appear either quite spontaneously or as a more or less distinct volley on insertion or movement of the needle electrode. Another type of potential, moreover, is often seen, known as sharply rising positive potentials (Jasper and Ballem, 1949; Kugelberg and Petersen, 1949). Both these types of activity provide important clinical information. They indicate that the muscle is denervated, with all that that implies in the prognosis.

Material and Methods

The subjects of this investigation comprised fourteen cases of ocular muscle palsies (Tables I and II) and ten cases of limitation of ocular movement of obscure origin, 24 cases in all. The majority were examined on two or more occasions, and in about half of the cases several muscles were studied. The time that elapsed between the onset and the first examination in the fourteen cases of palsy was less than 2 months, except in Case 7 when it was 10 months.

The investigation was carried out with the subject in the supine position. The patient was made to fix his gaze on a point of light which could be moved either along a tangent scale on the ceiling or along the arc of a perimeter. The zero point of the scale on the ceiling was placed perpendicularly above the eye and 230 cm. from it. The distance of the arc of the peripmeter from the eye was 35 cm.

The sound and paretic eyes were used alternately for fixation. When the paretic eye was used, the sound eye was covered. Great trouble was taken to get the patient to contract the muscle under examination to the greatest possible extent.

For determination of the position of the paretic eye the angle of deviation was measured, using the arc of the perimeter with a lamp moving along it. While the patient gazed with the sound eye at a small object about 3 m. away the angle of the paretic eye was measured using the corneal light reflex.

The method of recording the electrical activity has been described in earlier reports (Björk and Kugelberg, 1953a). Monopolar needle electrodes were used with the reference needle in the conjunctiva, bridge of the nose or lobe of the ear. To study the activity in the orbicularis oculi muscles a needle was inserted through the skin of the upper or lower eyelid.

Action potentials were amplified by differential amplifiers and the records made with a cathode ray tube and a moving paper camera. When only one amplifier was employed the second beam of the oscilloscope was used for time recording.

Results

(1) Lesion of Peripheral Motor Neuron

OCULOMOTOR PALSY.—Seven cases were examined (Table I, overleaf). They showed great similarity as regards aetiology, clinical picture, and course, as exemplified in Cases 1 and 6.

Case 1, male, aged 48, gave a history of sudden total paresis clinically of all external and internal muscles supplied by the 3rd nerve on the left side. Arteriography showed an aneurysm on the left internal carotid near the origin of the posterior communicating artery. About 6 weeks after the onset an operation was performed a silver clamp being placed on the stalk of the aneurysm. Two weeks later the eye was still unchanged (Fig. 1, overleaf). The eye could not be opened at all voluntarily. On gaze to the extreme right the eye stopped in a moderately abducted position.

An electromyogram was taken one week before the operation and again 2 weeks after it, on both occasions on the levator palpebrae and internal rectus muscles. No voluntary

TABLE I

CASES OF OCULOMOTOR PALSY

Case No.	Age (yrs)	Sex	Cause	Clinical Picture	Muscle Examined	Electromyographic Record on Maximal Contraction
1	48	M	Aneurysm of internal carotid	Left. Total paresis of all muscles	Levator palpebrae	No voluntary activity ? Fibrillations
					Interior rectus	
				Left. No change	Levator palpebrae	Voluntary activity A few potentials ? Fibrillations
					Internal rectus	
2	51	M	Aneurysm of internal carotid	Left. Total paresis of all external muscles	Levator palpebrae	No voluntary activity Probably fibrillations
					Internal rectus	
3	43	F	Aneurysm of internal carotid	Left. Paresis of all external muscles, in regression. Considerable ptosis on forward gaze	Levator palpebrae	Very moderate reduction of potentials In some places normal picture
				Further regression. Limited mobility of globe on upward gaze	Rectus superior	Voluntary activity A few potentials
4	66	F	Aneurysm of internal carotid	Left. Paresis in regression. Considerable ptosis on forward gaze	Levator palpebrae	Voluntary activity Moderate reduction in number of potentials
5	67	F	Hypertonia Cerebral injury	Right. Paresis in regression. Very moderate ptosis	Levator palpebrae	Voluntary activity In some places a very moderate reduction in number of potentials. Mainly normal picture
6	50	F	Skull fracture	Left. Total paresis of all muscles	Internal rectus	Voluntary activity One potential recruited
				Left. Possibly slight improvement in mobility of globe	Internal rectus	Voluntary activity Some potentials
7	69	M	Hypertonia	Left. Total paresis of all external muscles. On gaze to extreme right fixating with sound eye, left eye stops 10° outwards	Internal rectus	Voluntary activity A few potentials
				Left. Improvement. Eyelid can be raised a few mm. on maximum upward gaze	Levator palpebrae	Voluntary activity Moderate reduction in number of potentials

FIG. 1.—Case 1, man aged 48, clinical picture of total left oculomotor palsy. Two upper pictures show eye completely closed on gaze straight ahead and also on strong effort to raise lid when looking upwards. Two lower pictures show position of eye when sound eye looks straight forward and to extreme right respectively. Paretic eye directed outward and slightly downward does not move forward fully to midline. Pupil greatly dilated.

activity was demonstrated in the first study. The second revealed a distinct voluntary activity in these muscles. Thus on maximal contraction a small number of action potentials were mobilized in both muscles (Fig. 2). On observation at high sweep speed the frequency of two different potentials was calculated as 200 and 180 per sec. respectively.

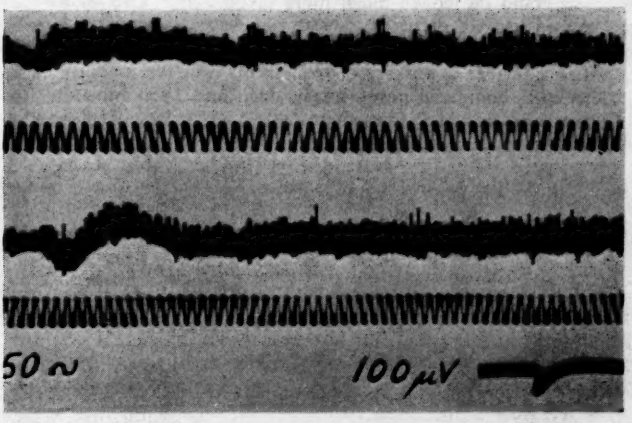

FIG. 2.—Case 1, record from levator palpebrae superioris muscle (upper tracing) and internal rectus (lower tracing) on paretic side. Activity at maximal contraction. Only a few potentials are recruited and these are of high frequency.

In addition some small potentials were observed which appeared to be independent of the voluntary activity and were thus suspected fibrillations.

The occurrence of voluntary activity in the later investigation indicates that an improvement had taken place. The prognosis could therefore be judged favourably, although the presumed presence of fibrillations gave reason to suspect that the recovery would

be slow. The subsequent course confirmed this. The mobility of the globe and eyelid increased slowly but continuously. Five weeks after the last electromyogram was taken the patient was able to raise the eyelid sufficiently to free the lower half of the pupil and the mobility of the globe to each side was fairly good. Three months later, *i.e.*, over 6 months after the onset, the condition was distinctly improved, but he was troubled by diplopia, particularly when looking far upwards or downwards.

Case 6, female, aged 50, had had a fracture of left temporal bone in a motor-cycle accident, followed immediately by a clinically total left-sided paresis of all muscles served by the 3rd nerve. Examination one month after the accident revealed the same clinical picture of total paresis of all muscles. On gaze to the extreme right the left eye stopped in a position of considerable outward deviation (Fig. 3).

FIG. 3.—Case 6, woman aged 50, clinical picture of total left oculomotor palsy. Pictures show the position of paretic eye when sound eye looks straight forward and to extreme right.

An electromyographic examination was then made of the internal rectus and in several different needle positions showed voluntary recruitment of an action potential which, on a maximum effort of contraction, reached a frequency of 200 per sec. (Fig. 4). This was not therefore a case of total paresis, as the clinical picture had given reason to suppose, and the prognosis could be judged much more favourably. Two weeks later a slight improvement seemed to have occurred, though not distinctly measurable. The electromyogram now showed that some potentials were being recruited, which indicated an improvement. After a further 2 weeks a distinct increase in the mobility of the eye was noticeable. There was continued improvement later, and 2½ months after the accident the eye could be moved up to 15° inwards.

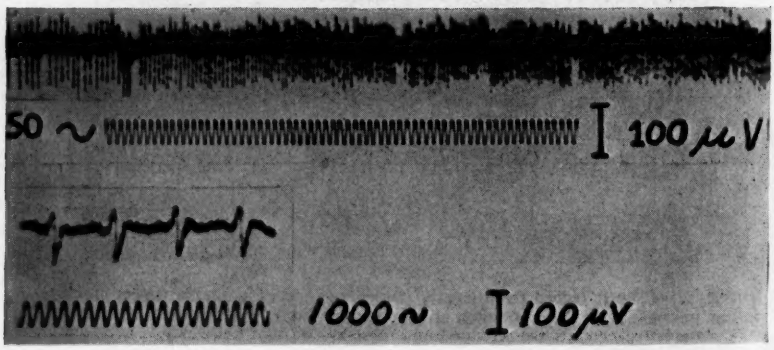

FIG. 4.—Case 6, record from internal rectus muscle on paretic side. An action potential is recruited which attains a high frequency on maximal contraction. Detail picture at high sweep speed shows a frequency of about 200/sec.

The typical electromyographic picture produced by partial injury was also seen

in Case 3 (Fig. 5). The eye manifested a limited mobility upwards, and the recorded activity in the rectus superior muscle is typical of a severe lesion of the peripheral motor neuron, *i.e.* a few action potentials of high frequency. In Fig. 5 (upper tracing) an uneven recruitment is noticeable. This occurred when the patient attempted fixation which demanded a powerful upward movement of the eye. She could not maintain an even activation of the available motor neurons. Several potentials of high frequency were mobilized, but after scarcely a tenth of a second one after the other fell out and the frequency of the individual potentials dropped, after which a stronger activation recommenced, and so on. Nor did the eye manifest any continuous fixation, but jerky movements of paretic nystagmus were observed. At times the eye could be kept still, and the activity was continuous with high frequency of the individual potentials (Fig. 5, lower tracing).

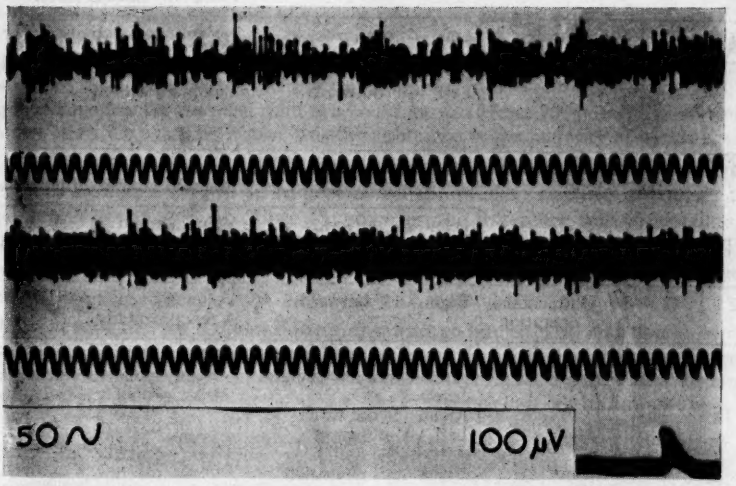

FIG. 5.—Case 3, record from paretic rectus superior muscle on strong effort to look upwards. Upper tracing shows uneven recruitment; this changes at intervals to more even recruitment (lower tracing).

Particularly as regards the levator palpebrae superioris, it is difficult to draw any conclusions from the clinical picture in respect of the real severity of the injury to the peripheral motor neuron. The position and mobility of the eyelid varied greatly with the fatigue of the patient and position of the head. A simultaneous paresis of the rectus superior, which was found in all cases, added further to the difficulty in diagnosis, as also did the patient's tendency actively to close one eye in order to avoid double images. Cases 3 and 4 are examples of considerable ptosis, which may interfere with vision on directly forward gaze, in spite of a very slight reduction in the number of potentials or even, at least during short periods, a normal number.

ABDUCENS PALSY.—Seven cases were studied (Table II, overleaf). The fact that these cases could be examined on several occasions made a more detailed analysis possible. Compared with the cases of oculomotor palsy, these cases presented a more varied aetiology and clinical course.

TABLE II
CASES OF ABDUCENS PALSY

Case No.	Age (yrs)	Sex	Cause	Position of Paretic Eye when Sound Eye fixates Farthest Inwards	Electromyographic Record from External Rectus Muscle on Maximal Contraction
8	53	F	Polyneuritis	Right. 10° inwards	Voluntary activity Moderate reduction in number of potentials
9	34	F	Unknown	Right. 15° outwards	Voluntary activity Reduced number of potentials
				Right. 30° outwards	Voluntary activity Practically normal picture
10	70	F	Diabetes mellitus	Left. 5° outwards	No voluntary activity Fibrillations
				Left. 10° outwards	Voluntary activity Greatly reduced number of potentials
11	59	M	Unknown	Left. 10° outwards	Voluntary activity Reduced number of potentials
				Left. 25° outwards	Voluntary activity No certain reduction in number of potentials
12	42	M	Intracranial operation	Right. 15° outwards	Voluntary activity A few potentials
13	57	M	Unknown	Bilateral paresis. (Fixation with contralateral eye). Right. 5° outwards Left. 5° outwards	Right. Voluntary activity. Greatly reduced number of potentials. Possibly fibrillations.
				Right. 15° outwards Left. 15° outwards	Right. Voluntary activity. Reduced number of potentials. In some places almost normal picture
				Right. 20–25° outwards Left. 15° outwards	Right. Voluntary activity. Normal picture
14	52	F	Unknown	Left. 5–10° inwards	Left. No voluntary activity.
				Left. Hardly 5° inwards	Left. Voluntary activity. One potential

The typical picture in lesions of the peripheral motor neuron was clearly demonstrated in this series also. The two following cases (13 and 8) are of special interest.

Case 13, male, aged 57, suddenly developed a bilateral abducens palsy. The eyes were capable of only about 5° outward movement (Fig. 6, opposite). Recordings from the external rectus muscle of the right eye showed that apparently only one potential was recruited on maximal contraction and that it attained a high frequency (Fig. 6, left recording). In another position of the needle a large, though definitely reduced, number of potentials

FIG. 6.—Case 13, record from paretic right external rectus on maximum effort to look to right. The two tracings represent two different sites of needle electrode; in first tracing only one potential is seen, in the other several.

were obtained (Fig. 6, right recording).

The right eye could move about 15° outwards 14 days later. There was now greater activity in the external rectus muscle. A rich supply of potentials was mobilized, so that sometimes an almost normal picture was obtained.

After a further 14 days the right eye could move at least 20° outwards. A generally normal electromyographic picture was then obtained in the majority of needle positions.

The fact that somewhat different pictures are obtained from different needle positions (which is known in respect of other muscles) applies, of course, to the ocular muscles as well. In Case 13, however, the difference was very striking and showed the necessity of investigations in a number of needle sites. On all three occasions an uneven recruitment was observed, but the picture was somewhat unlike that of Case 3. This was also well demonstrated in Case 8.

Case 8, female, aged 53, had polyneuritis with abducens palsy and flaccid paresis of extremities. The illness had started 10 months previously and no change of eye status has been revealed during the last 3 months. On direct forward gaze with the sound eye the paretic eye lay 30° inwards; on gaze to the extreme right it lay 10° inwards (Fig. 7).

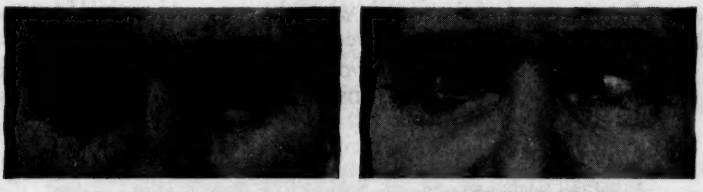

FIG. 7.—Case 8, woman aged 53, right abducens palsy. On directly forward gaze with sound eye, right paretic eye is directed 30° inwards. On gaze to extreme right, right eye is directed 10° inwards.

Electromyographic examinations on two different occasions gave concordant results (Fig. 8A, overleaf). When several potentials were recruited, the recruitment took place in such a way that for short periods the individual potentials showed a greater frequency and a tendency to group together. These small bursts, which arrived rather irregularly, were separated by periods of recruitment of only a few single potentials of low frequency.

This picture of uneven recruitment of action potentials which was seen in the above case and also in Case 13 was very characteristic and constant; it was found in most needle positions, and appeared on very moderate effort of contraction. On greater contraction so rich a supply of potentials was recruited that the entire baseline became covered and the characteristic grouping became less and less pronounced.

In addition to investigations of the number of potentials and their manner of recruitment, observations were made on the size and form of the potentials and the presence of fibrillations.

(*i*) *Size and Form of Potentials.*—The form and size of the individual potentials was in most cases similar to that seen in non-paretic eye muscles. A detailed analysis was not always made. In a few cases there were some striking divergences. Thus, in Case 8, a number of polycyclic potentials were seen, one of which was very broad and of complicated shape (Fig. 8D).

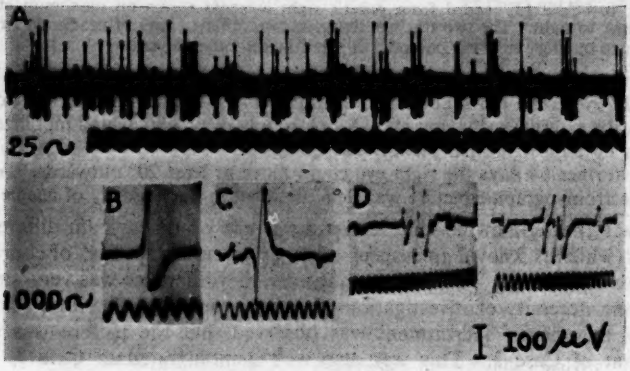

FIG. 8.—Case 8, record from paretic external rectus. On moderate contraction an uneven recruitment is obtained (A). Detail pictures at high sweep speed show that several potentials are of unusually long duration (B and C) or polycyclic (D).

Similar potentials were observed in Case 9, the first examination being made one week after the onset of the paresis when the eye could move 15° outwards, and the last, 11 days later, when it moved 30° outwards. On the latter occasion (Fig. 9) the activity was practically normal. A detailed analysis at high sweep speed (Fig. 9, lower tracing), however, showed a number of polycyclic

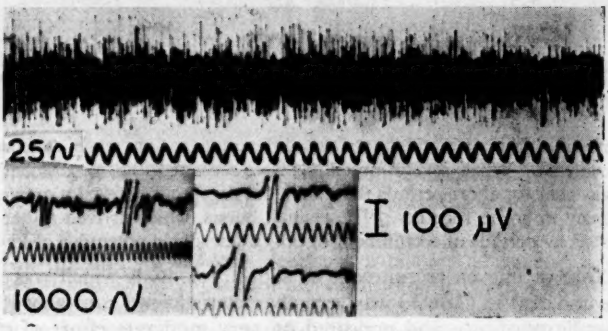

FIG. 9.—Case 9, record from paretic external rectus on maximal contraction (upper tracing) showing abundance of potentials. Detail pictures at high sweep speed (lower tracings) show a few polycyclic potentials.

potentials. Polycyclic potentials were also observed at the first examination. In Case 13, likewise, several very broad polycyclic potentials were seen.

It has long been known from other muscles (Weddell and others, 1944) that an abundance of polycyclic action potentials is found chiefly on regeneration of the nerve fibres.

The possible occurrence of fibrillations and the clinical course are naturally significant factors in assessing whether a regeneration exists. In Case 8 the paresis had already been present for ten months when the first examination was made. No fibrillations were found, but it is of interest that they were demonstrated in several of the extremity muscles. In Case 13, with bilateral abducens palsy, fibrillations may possibly have existed. The restitution was fairly rapid and a clinical improvement was noted after only three weeks. Case 9 manifested an improvement in mobility after about one week and the regression was rapid throughout.

Whether or not reinnervation took place in these cases is, of course, difficult to decide. Perhaps a reinnervation occurs very rapidly in the ocular muscles.

No certain explanation can be given, either, of the strikingly large potentials observed in two of the cases. Potentials of abnormally long duration, without being polycyclic, were demonstrated in conjunction with polycyclic potentials in Case 8 (Figs 8 B and C), several of them having a duration of about 3 m.sec. Similar potentials were also seen in Case 11. Potentials of this kind may be associated with a picture of regeneration. In chronic nerve lesions it is known that in other muscles potentials of high amplitude (Denny-Brown and Pennybacker, 1938; Kugelberg 1947, 1949; Yahr and others, 1950) and of long duration (Buchtal and Clemmesen, 1941) appear.

(*ii*) *Fibrillations.*—These are difficult to recognize because the ocular muscles cannot with certainty be relaxed to the point where all voluntary activity disappears. Their recognition is also difficult since their appearance is similar to the voluntary potentials of the ocular muscles. In Case 10, however, fibrillations were observed with certainty. The patient, a 73-year-old woman, was examined 3 weeks after the onset of the paresis. The eye could not move more than 5° outwards, and no voluntary activity was found during the electromyographic examination. On the other hand, small potentials were seen (Fig. 10, overleaf) which were independent of the position and movements of the eye. They had an irregular, slow frequency, and lasted uninterruptedly for over half an hour. Ten days later the picture was clinically unchanged. The electromyographic examination showed fibrillations, though in less profusion than earlier. Voluntary activity was demonstrable seven weeks after the onset. An improvement in mobility was noticed at the same time, and after 4 to 5 months the mobility was practically normal.

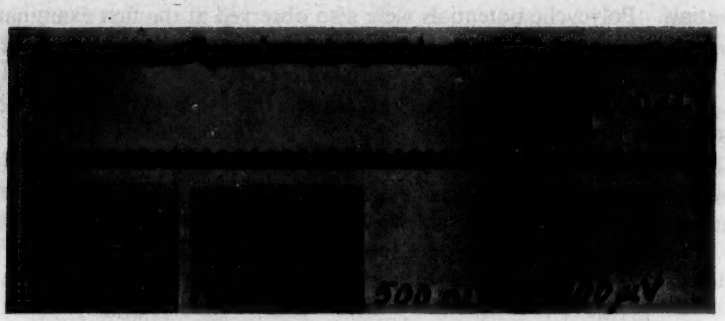

FIG. 10.—Case 10, record from paretic external rectus. Upper tracing shows fibrillations. Lower tracing shows detail picture at high sweep speed with spikes of about 85 μ V amplitude and 0·5 m.sec. duration.

In Case 2 an activity was observed that was probably due to fibrillations. Activity highly suspicious of fibrillations was also observed in Case 1, as mentioned above.

The presence of fibrillations indicates that, if restitution occurs, it will proceed slowly; this is confirmed by the three cases referred to.

Mobility of Paretic Eye and Electromyographic Findings.—To assess the degree of the paresis by ordinary clinical methods is easier in abducens palsy, as only one muscle is paralysed, than in oculomotor palsy. A comparison between the mobility of the paretic eye and the electromyographic findings in abducens palsy can be made from the data in Table II.

The limitation in the outward movement of the eye, which characterizes a total paresis, varies from case to case within quite wide limits. This is, for example, apparent in Cases 8 and 10. In Case 10 the eye could be moved 5° outwards and no voluntary activity was recorded electromyographically from the external rectus, while in Case 8 the eye stopped 10° inwards in spite of a quite considerable voluntary activity. It follows therefore that the normal clinical methods are inadequate for determining whether a paresis is total or not.

The greater the capacity of the eye to move outwards, the more certain it is, of course, that a voluntary activity will be found in the external rectus. Activity has also been demonstrated in the cases in which the eye could be moved 10° outwards or more.

In oculomotor palsy a determination of the severity of the paresis from the mobility of the eye appears to be even more problematical. In Case 6, as already mentioned, the paresis appeared to be clinically total in all extrinsic muscles. On maximum movement in the direction of the sound eye, the paretic eye stopped at 25° outward deviation. In spite of this manifestly poor mobility voluntary activity was not lacking in the internal rectus.

In clinically mild or moderately pronounced pareses it often happens that no definite reduction in the number of potentials is noticeable on myographic examination. Examples of this are Cases 9 and 11, in which a clinically manifest abducens paresis was present and the paretic eye could be moved 30° and 25° outwards respectively.

In some cases of loss of voluntary activity in the external rectus, the activity has been examined in the sound antagonist as well, that is in the internal rectus. It was found that, when the gaze was in the direction of the paretic muscle, a successive diminution of activity took place in the internal rectus, roughly the same as if the eye performed its normal movement. This observation is of help in the understanding of the reciprocal innervation of the ocular muscles, which will be dealt with in greater detail in a later paper.

(2) Mechanical Factors impeding Movement of the Eye

A dislocation of the globe due, for example, to trauma or to space-occupying lesions in the orbit may cause a limitation in its movement that simulates or conceals an eye muscle paresis. Two cases have been examined in which the globe was dislocated by atrophy of the orbital fat following trauma; in one case this was combined with a fracture that caused *inter alia* a lowering of the floor of the orbit.

Case 15, male, aged 51, gave a history of trauma in the right orbital region, without bone fracture or damage to the globe, which caused within the course of a few months an atrophy of the orbital fat, whereby the globe was dislocated inwards and downwards towards the floor of the orbit. The movement of the globe was limited, particularly upwards. On gaze straight ahead and fixation with the left eye, the right eye was directed 5° downwards. On raising the gaze to the upward maximum, the right eye moved very little and stopped at a maximum of 10° upwards (Fig. 11). On downward gaze there was no restriction of mobility. Laterally there was good mobility, but a slight limitation of outward movement.

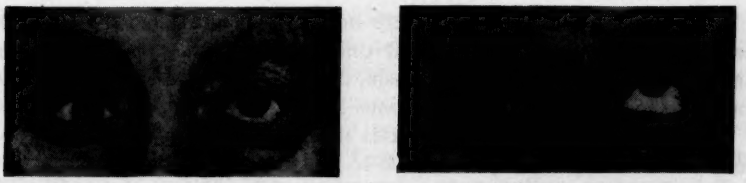

FIG. 11.—Case 15, male aged 51, dislocation of right eyeball following trauma. On gaze straight ahead with left eye, right eye is directed slightly downwards. On extreme upward gaze, right eye is directed 5–10° upwards.

An electromyographic recording from the rectus superior of the right eye showed no signs of lesion of the peripheral motor neuron. On fixation with the left eye on an object which moved from below upwards until the patient could no longer see it, the electromyogram from the right eye showed a continuously increasing activity, apparently quite independent of the limitation of movement—roughly as though the recording had been taken from the sound eye. When the right eye was directed straight ahead, an

abundance of potentials was observed (Fig. 12B). A considerable increase was seen on maximum upward gaze (Fig. 12C). Between these positions the right eye moved 5–10°.

During examinations of the superior rectus care must be taken to avoid the error of recording instead from the levator palpebrae superioris. This possibility was excluded by examining the activity during blinking as well. Simultaneous recordings were taken from the orbicularis oculi with the needle in the lower eyelid (Fig. 12A). The recording revealed a short increase in activity in connection with blinking. This cannot be referable to the levator, since in the latter muscle a strong momentary inhibition of the activity takes place on blinking (Björk and Kugelberg, 1953b).

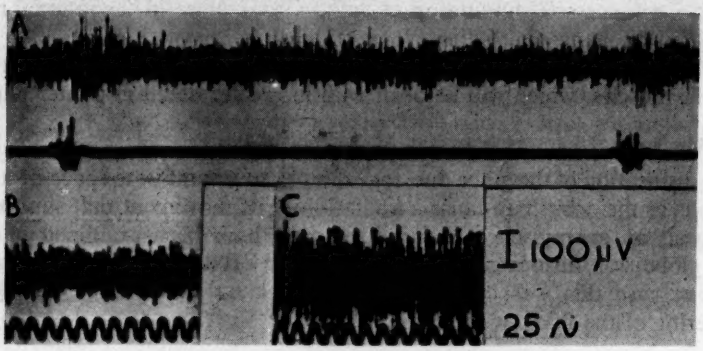

FIG. 12.—Same case as shown in Fig. 11.
(A) Simultaneous recording from right rectus superior (upper tracing) and from orbicularis oculi with needle in right lower eyelid (lower tracing). During blinking a momentary increase in activity takes place in rectus superior.
(B) Activity in rectus superior when right eye is directed straight ahead.
(C) Considerably increased activity, on maximal upward gaze. Fixation with sound eye in both instances.

The examination showed that there could be no severe injury of the peripheral motor neuron. This leads one to assume that the dislocation of the globe was the main cause of limited movement. The second case examined manifested identical conditions in all essential respects.

Limitations of movement that are innate or acquired in early childhood form a quite common group, but one that presents difficulty in clinical diagnosis. It includes nuclear aplasia, defective development of the muscles, abnormal tendon attachments, various kinds of childbirth lesion, and so on.

The cases present many difficulties in electromyographic examination as well. Technical problems arise due to the often abnormal topography and extreme thinness of the muscles. In some cases, however, an electromyographic study has been carried out successfully. For example, in two cases in which a total paresis was supposed, a good activity was revealed electromyographically.

Discussion

The method employed for the solution of a number of questions concerning the electrophysiology of ocular muscles (Björk, 1952; Björk and Kugelberg, 1953 a, b) has proved applicable, too, to the examination of pathological cases.

To study a paretic or suspected paretic muscle, an examination must be made in several different needle sites. This is of particular importance in myopathies and in denervation. The anatomical conditions are such, however, that certain portions of the ocular muscles are inaccessible, and at times it is difficult to obtain a suitable needle position at all. As it is not known exactly where in the muscle the point of the needle lies, it is probable that recordings are on many occasions taken from superficial positions or within the junction between the tendon and the belly of the muscle involving a very small pick-up range for the needle. This may possibly be the explanation why different sites have at times manifested a strikingly large difference in the number of action potentials that can be mobilized. Thus the limitations attaching to the method itself imply certain limitations in the possibilities of diagnosis.

The fundamental features in the electromyographic picture characteristic of a lesion of the peripheral motor neuron are also found in respect to the ocular muscles. The investigation has shown that a reduction in the number of potentials on voluntary contraction follows essentially the same pattern as in other muscles, but that it is impossible to demonstrate this reduction in cases of pareses of only moderate clinical severity. The explanation lies in the fact that the ocular muscles bring into play a particularly abundant quantity of potentials, so that, despite the disappearance of many of them, pictures manifesting a profusion of interfering potentials are still seen.

An uneven recruitment is often seen in a paretic eye muscle and may behave electromyographically in different ways. Thus on even slight contraction, involving the recruitment of few potentials, it is seen that they appear in small bursts between intervals of more sparse recruitment. With a rising effort of contraction the picture can be followed as long as not too many potentials are mobilized, when the picture becomes less characteristic.

Another picture may also occur on strong contraction, particularly when the number of potentials is greatly reduced: rapid recruitment of a number of potentials of high frequency, gradual diminution in frequency and disappearance of potentials, so that the activity sometimes falls to zero, again rapid recruitment, and so on. These pictures may vary considerably in different cases and on different occasions.

In uneven recruitment an ordinary inspection will normally reveal that the eye makes small jerky movements. These movements are observed still more clearly if, in addition to electromyographic examination, some detail in the fundus is examined through an ophthalmoscope. It is thus a question of paretic nystagmus. This may appear in various forms, both clinically and electromyographically, and will be analysed more closely in connection with nystagmus (to be published).

The rapid fatigue which distinguishes a palsied muscle is admirably revealed by electromyographic examination of paretic eye muscles, and possibly paretic nystagmus is also one of its manifestations. A remarkably

vigorous electrical activity can be obtained from a sudden effort of contraction, but the potentials can only be mobilized momentarily. In fairly pronounced pareses, therefore, there may be short periods in which the pictures appear quite normal. A vigorous effort of contraction is most conveniently obtained by covering the sound eye and getting the patient to fixate with the paretic eye. The effort to follow an object with the gaze within the area of activity of the paretic muscle mobilizes an innervation impulse which is stronger than that produced by fixation with the sound eye in its extreme positions.

As has been shown earlier (Björk and Kugelberg, 1953a), very high frequencies of the remaining potentials may be observed in paretic eye muscles, often up to about 200 per second. This was confirmed in the cases now investigated. The demonstration of high frequencies is significant as evidence that a vigorous effort of contraction has taken place. In eye muscle pareses a good check on the effort applied is also obtainable by observation of the position and movements of the sound eye. It has at times proved difficult or impossible to obtain high frequencies in spite of an apparently vigorous effort of contraction. This fact is known from severe pareses in other muscles and is probably due to failure of proprioception from the paretic muscle (Kugelberg, 1953).

Fibrillations are difficult to demonstrate in ocular muscles (Björk, 1952). The eye muscle potentials are normally of about the same appearance as fibrillations, and therefore other criteria must be sought, in particular that the fibrillations shall be independent of the voluntary contraction. Since complete voluntary relaxation of an eye muscle is not possible to achieve with certainty, it is mainly in cases of entire absence of voluntary activity that we are enabled to find fibrillations. Also, since the whole muscle is not accessible to examination, it may happen that fibrillations are not discovered. These difficulties explain the uncertainty in diagnosing some of the cases reported with respect to denervation and reinnervation.

All methods employed hitherto for assessing the severity of an eye muscle paresis and following its course are based on recording of the position and movements of the eye. The present investigation has shown that, in the assessment particularly of severe pareses, the electromyographic study can provide information that cannot be obtained by other methods.

When the limitation of movement in a paresis is of great severity, the picture of total paresis is encountered. The degree of limitation to be expected is apparent in the remarks of Cogan (1948):

> *Oculomotor Palsy.*—When the non-paralysed eye is fixating straight ahead, the paralysed eye is held in a position of divergence but can be brought to the midline by directing the gaze to the side opposite the paralysis.
>
> *Abducens Palsy.*—Convergent strabism with inability to turn the eye lateralward beyond the midline.

The electromyographic examination gives us exact information concerning the voluntary activity. When we relate this information to the clinical

picture, we find that voluntary activity is often manifested where the clinical picture suggested a diagnosis of total paresis. This is perhaps particularly striking in the oculomotor palsies. Thus in one case a distinct voluntary activity was demonstrated in the internal rectus muscle although the eye could not reach anywhere near the midline but stopped at 25° outward deviation on maximum outward fixation with the sound eye.

The course of severe pareses can be very accurately followed by electromyographic means. The number of action potentials is in these cases greatly reduced, and a slight alteration will therefore be readily noticeable. Such an alteration may occur without any change in the clinical picture. The action potentials of the ocular muscles are, as was shown earlier, small and must be assumed to represent weak motor units. This would seem to apply to a very marked extent to the small potentials which may appear at the beginning of a reinnervation.

The position assumed by the paretic eye depends upon many factors. It has been shown earlier that in normal cases, when the eye is in the primary position the motor unit activity is very considerable in all the recti (Björk and Kugelberg, 1953b). It is therefore natural that, in paresis of one or more muscles, the eyeball is strongly pulled in the direction of the sound antagonists. Other factors may also affect the dislocation; it will be sufficient here to mention the significance of a possible latent strabism which is manifested on disturbance of the binocular vision.

Limitation of the movement of the eye may be brought about, not only by neurogenic pareses, but also by other causes such as muscular affections and purely mechanical factors. Oedema and bleeding in the eyelid and orbit are examples of this, as is also a dislocation of the eyeball in connection with traumata of the orbital region. Other examples comprise congenital disturbances involving under-development of the muscles, abnormal tendon attachments, etc.

The investigation has shown that electromyography is an aid in differential diagnosis between these aetiologically quite distinct conditions.

Summary

An electromyographic investigation was made of ocular muscles in 24 patients with limited mobility of the eyeball, including fourteen cases of typical eye muscle palsy and two cases in which the main cause was assumed to be due to mechanical factors. In the remainder of the cases the cause was less distinct.

(1) Lesion of Peripheral Motor Neuron

(*a*) *Number and Form of Action Potentials.*—A reduction in the number of action potentials has been observed, the chief features of which accorded with experience from other muscles.

The duration and form of the action potentials seemed in most cases to be

the same as in non-paretic eye muscles. Some divergences have been observed, *e.g.* an abundance of polycyclic action potentials in three cases.

(*b*) *Fibrillations.*—This important sign also occurs in the ocular muscles and was definitely observed in one case and appeared probable in two others. Since they have the same appearance as the normal action potentials and complete voluntary relaxation of an eye muscle is not possible to achieve with certainty, they are difficult to diagnose.

(*c*) *Uneven Activation.*—In a number of the cases the recordings showed an uneven activation of the residual action potentials of characteristic appearance. At the same time small jerkings of the eyeball were observed, so called paretic nystagmus.

(*d*) *Paresis.*—By aid of electromyography it is possible to decide whether an eye muscle paresis is total. In this respect the clinical methods of examination are unsatisfactory. It was thus shown that in cases of paresis, which were clinically diagnosed as total, a voluntary activity was manifested in the electromyographic examination.

The course of the severe pareses can be followed with great accuracy electromyographically, for, as long as the number of action potentials is fairly small, even very slight changes are easily noticeable. These small changes often occur without any change at all in the clinical picture.

In moderately pronounced or mild cases of paresis no reduction in the number of action potentials is as a rule observed by the methods used here.

(2) Mechanical Factors causing Limitation of Movement

In two cases involving change of the anatomical conditions in the orbit following trauma, it was shown that electromyography assists in differentiating between ocular muscle paresis and limitation of movement due to mechanical factors.

In some indistinct cases, as in congenital limitation of movement, electromyographic examination can be of aid in diagnosis.

REFERENCES

Björk, Å. (1952). *Experientia*, **8**, 226.
———, and Kugelberg, E. (1953a). *Electroenceph. clin. Neurophysiol.*, **5**, 271.
———, ——— (1953b). *Ibid.*, **5**, 595.
Buchthal, F., and Clemmesen, S. (1941). *Acta psychiat. neurol. (Kbh.)*, **16**, 143.
Cogan, D. G. (1948). "Neurology of the Ocular Muscles". Thomas, Springfield, Ill.
Denny-Brown, D., and Pennybacker, J. B. (1938). *Brain*, **61**, 311.
Jasper, H., and Ballem, G. (1949). *J. Neurophysiol.*, **12**, 231.
Kugelberg, E. (1947). *J. Neurol. Neurosurg. Psychiat.*, **10**, 122.
——— (1949). *Ibid.*, **12**, 129.
——— (1953). Personal communication.
———, and Petersen, I. (1949). *J. Neurol. Neurosurg. Psychiat.*, **12**, 268.
Lindsley, D. B. (1935). *Amer. J. Physiol.*, **114**, 90.
Seyffarth, H. (1938). *Acta psychiat. neurol. (Kbh.)*, **13**, 297.
——— (1940). *Skr. norske Vidensk.-Akad., I. Mat.-nat. Kl.*, No. 4.
Weddell, G., Feinstein, B., and Pattle, R. E. (1944). *Brain*, **67**, 178.
Yahr, M. D., Herz, E., Moldaver, J., and Grundfest, H. (1950). *Arch. Neurol. Psychiat. (Chicago)*, **63**, 728.

Brit. J. Ophthal. (1954) **38**, 545.

DEGENERATIONS OF THE DOG RETINA*

V. GENERALIZED PROGRESSIVE ATROPHY OF UNCERTAIN AETIOLOGY

BY

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DURING an investigation of a hereditary retinal atrophy of Red Irish Setter dogs (Parry, 1953b), we encountered syndromes which were somewhat similar to the disease observed in Irish Setters but in which evidence of a similar hereditary origin, or of distemper-complex virus infections (Parry, 1954a), or glaucoma (Parry, 1953c) were lacking. In these cases there was a generalized degeneration of the retina and an atrophy of the pigment epithelium as in the hereditary disease.

This paper reports the findings in three cases in which we were able to carry out detailed clinical and pathological observations. The cases were of two types: first, a familial type in which two of three litter mates were affected, and secondly, an idiopathic type with no pertinent medical history.

Methods and Materials

The methods and terminology have been described in previous papers (Parry, 1953a, b, c). The cases described were natural ones. The first two were kept in isolation kennels under observation for up to 2 years and the test-matings were carried out under controlled conditions (Parry, 1953b). The electroretinograms were recorded by the team (Parry, Tansley, and Thomson, 1953).

Results

(1) Familial Type

The dogs affected each weighing about 17 kg., came from a litter of three male pedigree Afghan Hounds. They had been vaccinated against Laidlaw-Dunkin distemper in adolescence and had no pertinent medical history of febrile illness, etc. No defects of vision were noted until they were 1½ years old.

Case 1 (DO 61).

Clinical Data.—This dog was noticed to be night blind when 18 months old. He was examined at 2 years old, when he entered the research kennels. At this time the dog was completely day and night blind in both eyes; the average pupil size was 13/15 mm., the pupillary light reactions and eye preservation reflex were absent; the retinal blood vessels were reduced to a single small dorsal vessel; the tapetal reflection was a very bright, light green, and the fundus texture was crystalline rather than granular with very few stellulae of Winslow; the non-tapetal fundus was orange brown; the lenses were clear and there was no increased ocular tension. The retina of the left eye became detached 3 months later, but the outcome could not be observed owing to the development of a posterior capsular cataract. There was no further significant change until the dog was destroyed when 4½ years old, 2 years after the development of complete blindness, by the intravital injection of Kolmer's cold-blood fluid under deep pentobarbital sodium anaesthesia.

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Morbid Anatomy.—The autopsy revealed no pertinent abnormalities. The eyes were normal when opened.

Histology.—The findings were indistinguishable from a case of Stage 3b of the hereditary atrophy of Red Irish Setters (Fig. 1).

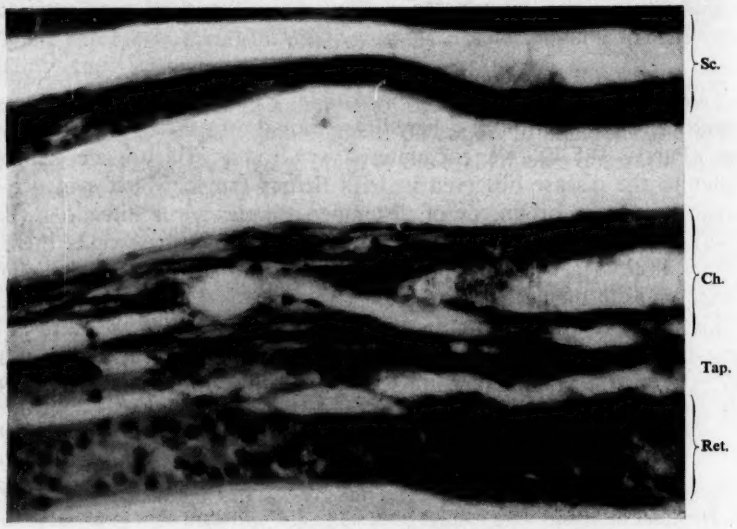

FIG. 1.—Section of retina of a dog with familial generalized progressive atrophy (Case 1); equatorial tapetal fundus. The retina is reduced to a narrow sclerosed band, and the pigment epithelium has disappeared. Note the advanced atrophy of the choroid. Intravital fixation with Kolmer's cold-blooded fluid. Heidenhain's iron haemotoxylin (× 270).

The following system of labelling the retinal structures applies to all the figures:

Sc. Sclera
Ch. Choroid
Tap. Tapetum
C.C. Chorio-capillaris
Ret. Retina
1. Pigment epithelium
2. Rod and cone layer
3. External limiting membrane
4. Outer nuclear layer
5. Outer fibre layer
6. Inner nuclear layer
7. Inner fibre layer
8. Ganglion cell layer
9. Optic nerve fibres
10. Internal limiting membrane

Case 2 (DO 165).

Clinical Data.—This dog was said to have had normal day and night vision until it was 4 years old. When examined at 4¼ years old, it was completely day and night blind; the average pupil size was 13/14 mm.; there were early small focal nuclear cataracts in both lenses; the pupillary light and eye preservation reflexes were absent; the fundi were similar to Case 1 with very few blood vessels visible and the papillae were apparently completely avascular. The dog was destroyed when 5 years old, at least one year after the blindness had developed, the same method being used as in Case 1.

Morbid Anatomy.—The autopsy revealed no pertinent abnormalities and the eyes were normal when opened.

Histology.—The sections showed advanced sclerosis as in Case 1, but remains of the retina were beaded due to the complete disappearance of the retina in some areas while in others some layer organization remained.

The third dog of the same litter was examined with Case 1 when 2½ years old; at that time it had normal day and night vision although the test for night vision was carried out on a clear night with a full moon; the average pupil size was 11/14 mm.; the pupillary light reactions were fairly brisk. Over the tapetal fundus the reflection, blood vessels, and stellulae of Winslow were normal, although the texture was slightly crystalline. The non-tapetal fundus was normal, apart from an oval grey fundus defect 5×8 mm. (apparent size) immediately ventral to the papilla. At 4¼ years old this dog was still said to have perfect sight; there has been no opportunity for another examination.

The general health of all three dogs was excellent and there were no signs of defects of the nervous system other than the blindness.

Test-Matings with Red Irish Setters carrying the Gene for Hereditary Generalized Progressive Retinal Atrophy.—As the clinical observations suggested that the Afghan Hounds might well be suffering from the hereditary atrophy of Red Irish Setters, several matings were carried out in the hope of determining whether or not this syndrome in Afghan Hounds was determined by a similar genetic factor. As no blind or closely related female Afghan Hounds were available, the dogs were mated with Red Irish Setter bitches whose genotypic constitution relative to the retinal atrophy was known from other studies (Parry, 1953b). The results are given in the Table. The diagnosis in the first two litters was based on clinical, electrophysiological, and pathological examinations; in the third litter on clinical and electrophysiological data only. As all the offspring were normal, we conclude that the blindness in the Afghan Hounds was not determined by the gene responsible for the progressive retinal atrophy in Red Irish setters.

Comment.—This type of progressive retinal atrophy is characterized by a familial incidence, the development of complete blindness by middle life without other signs of ill-health and a symmetrical degeneration of the retina and pigment epithelium without pigment dystrophy, which is very similar histologically in its advanced stages to the hereditary disease of Red Irish Setters.

TABLE

RESULTS OF MATING TWO AFGHAN HOUNDS WITH RED IRISH SETTER BITCHES CARRYING THE GENE FOR GENERALIZED RETINAL ATROPHY

Sire	Dam		Offspring		
	Genotype	No.	Total	♂ ♀	Affected
Afghan Dog DO 61	Carrier R.I.S.	DO 19	10	5 5	0
Afghan Dog DO 61	Affected R.I.S.	DO 6	9	5 4	0
Afghan Dog DO 165	Affected R.I.S.	DO 23	7	5 2	0

(2) IDIOPATHIC TYPE

In the one case of this type there was no evidence of glaucoma or of damage to the nervous system suggesting a prior distemper-complex infection and related animals were said to have normal vision.

Case 3 (Yellow Labrador Retriever Bitch)

Clinical Data.—This animal was aged 8 years, and weighed about 27 kg.; she was said to have gone blind suddenly 3–4 weeks previously without any other illness. Both eyes were found to be affected similarly; day vision was still present, but she was completely

night blind. The average pupil size was 8/15 mm.; the ocular tension was normal; the eye preservation reflex was absent but very slight pupillary light reflexes were still present; the lens showed some refractive errors without discrete opacities. In the fundus the retinal blood vessels were markedly reduced, only the main branches remaining; the tapetal fundus was a greenish orange with a crystalline texture. The blood vessels of the papilla, which was a pale beige colour, were reduced to $\frac{1}{3}$ to $\frac{1}{4}$ of their normal size. Both eyes were affected similarly.

A year later the dog was almost completely day blind; the average pupil size was 10/15 mm.; the pupillary light reflexes were absent on the left and only just present on the right; there were early nuclear cataracts in both lenses. A year later, when at the age of 10 years the dog was destroyed, the same method being used as in Case 1, the nuclear cataracts were slightly denser and there were signs of an iridocyclitis with adhesions of the iris to the lens capsule in the right eye, which had not been observed previously.

It was not possible to trace the litter mates of this bitch but they were said to have shown no signs of defective vision during their working life as gun-dogs. A daughter, examined at 2 and 3 years old, was normal, and no signs of defective vision in her litter mates had been reported to the breeder.

Morbid Anatomy.—When the right eye was opened the lens capsule was found to be adherent to the vitreous humour, which was more opaque than usual and adherent to

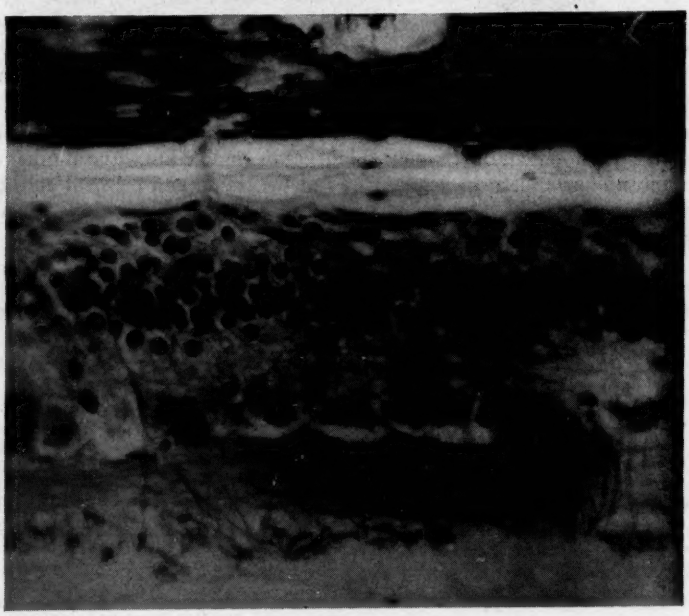

FIG. 2.—Section of retina of dog with idiopathic generalized progressive atrophy (Case 3); peripapillary tapetal fundus. The pigment epithelium, rod and cone layers, and outer nuclear layers have disintegrated. The inner nuclear, inner fibre, and optic nerve fibre layers are relatively intact, but the nuclei of Muller's cells are prominent. The large ganglion cells are still present, although they are degenerating, and many " ghost " forms can be seen. Note the rounded, deeply-pigmented cells in the choroid. Intravital fixation with Kolmer's cold-blooded fluid. Heidenhain's iron haematoxylin. (× 270).

the retina. The left eye was similar, but the abnormalities were less marked. The optic nerves and brain showed no macroscopic lesions. There were no significant lesions in the other organs.

Histology.—The retina was similar in both eyes and showed advanced degeneration and sclerosis over both non-tapetal and tapetal portions of the fundus, the central tapetal fundus alone retaining signs of the layer organization.

Over the whole fundus the rods and cones had disappeared. The pigment epithelium and chorio-capillaris were greatly reduced or missing, so that the external limiting membrane was adjacent to the choroid over most of the fundus, although in a few places next to large trans-tapetal capillaries there were foci 20–40μ long, in which pigment epithelial cells remained alongside enlarged capillaries of the chorio-capillaris; these cells contained round brown pigment granules mostly unstained by azo-carmine. The remaining inner retinal layers were reduced to about 60μ thick over the non-tapetal fundus, with complete loss of the layer organization and reduction of nuclei to a few lying in a sclerosed matrix with occasional large round pigment cells of choroidal type. The sclerosis was not quite as advanced over the peripheral tapetal fundus, the retina being about 100μ wide. However, in the central tapetal fundus, the layers were still present (Fig. 2). Here in the peripapillary zone the outer nuclear layer was reduced to a single interrupted layer of nuclei, but the inner nuclear layer, ganglion cells, and optic nerve fibres were still present, although degenerate. In the mid-tapetal fundus the inner nuclear layer was reduced to 2–3 nuclei thick, only an occasional ganglion cell remained, and the nerve fibre layer had almost disappeared.

The larger retinal blood vessels were still present but buried deeply and with very marked thickening of their walls (Fig. 3); at one point a large vessel had anastomosed with the

FIG. 3.—Same preparation as Fig. 2; mid-tapetal fundus. Note the two small blood vessels with severely fibrosed walls lying in the sclerosed remains of the retina. Azo-carmine. (× 870).

chorio-capillaris. The tapetum was not markedly abnormal apart from the walls of the trans-tapetal capillaries which were thickened. The choroid was much reduced, being only about 100 μ wide, and the media of the larger arteries near the papilla showed a marked regular thickening due to swelling of the cells (Fig. 4). The normally elongated choroidal pigmented cells had become rounded and in a few places some of these cells were lying against or just within the outer margin of the retina over the non-tapetal fundus. The optic papilla was vacuolated with fragmented nerve fibres, and the adjacent orbital optic nerve showed advanced degeneration.

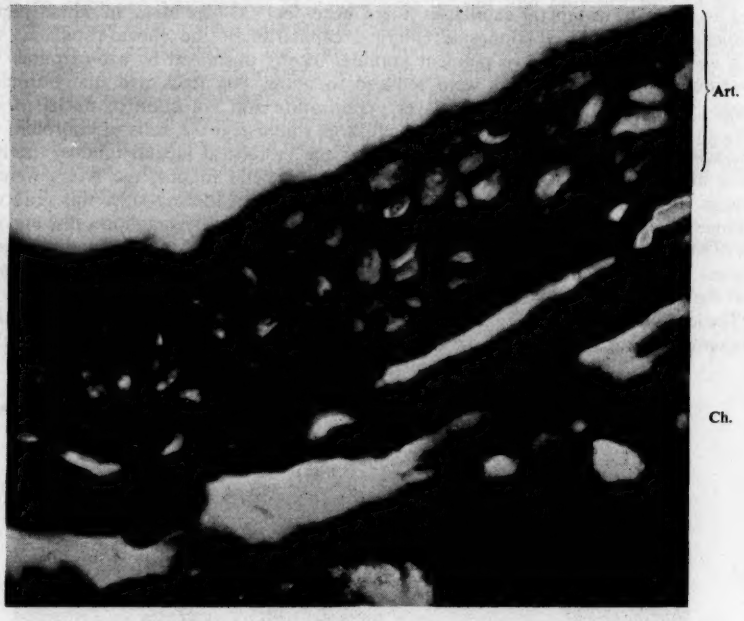

FIG. 4.—Section of choroid from the case illustrated in Figs 2 and 3; peripapillary tapetal fundus. Note the wall of an artery (Art.) showing great thickening due to cellular proliferation. Azo-carmine. (× 870).

Comment.—In this idiopathic type of generalized progressive atrophy, which appears late in life, the degeneration affects the pigment epithelium and the outer layers of the retina before the inner, and is least advanced over the peripapillary tapetal fundus, the peripheral fundus being much more severely affected. There are severe degenerative changes in the walls of the retinal and choroidal blood vessels.

Discussion

Although the data available regarding these two types of generalized progressive retinal atrophy are insufficient to establish their aetiology and the stages of their development, these cases are important as illustrations of syndromes which may be confused with the hereditary condition and must be excluded when diagnosing the latter.

The familial type in Afghan Hounds is of particular interest in this connection, for the disease appeared to be very similar to that of Red Irish Setters, even to the late development of cataracts, the only suspicious gaps in the clinical history being the absence of evidence of night blindness in infancy and adolescence and the relatively late onset of day blindness. The matings with Red Irish Setters showed conclusively that the Afghans were not carrying the gene for the Irish Setter disease in a homozygous form and probably not at all, and that, therefore, they were not suffering from the same hereditary syndrome. We cannot rule out the possibility of another form of inherited retinal atrophy controlled by genes different from that responsible for the disease in Red Irish Setters. However, more evidence will be required before such an assumption is justified. In the meantime we may exclude certain syndromes of retinal degeneration of which these two cases might be considered to be a late stage, namely post-distemper retinopathy and central progressive atrophy (Parry, 1954b), by the absence of pigment epithelial cell dystrophy, and we may exclude the atrophy secondary to glaucoma by the absence of clinical evidence of glaucoma and the symmetrical distribution of the degeneration.

The detachment of the retina observed in one case is an uncommon event in the dog and we have not seen it in any cases of the hereditary syndrome.

In the single case of the idiopathic type the well-marked degenerative changes in the walls of the retinal and choroidal blood vessels are similar to those seen in distemper and post-glaucomatous retinopathies, but the absence of pigment epithelial dystrophy and the relative sparing of the ganglion cells make previous distemper-complex infection and glaucoma unlikely causes of the degeneration. However, the varying severity of the lesions in different parts of the fundus, with the peripapillary tapetal fundus least affected, and the greater damage to the first-order neurones and optic nerve fibres, is similar to that seen in the distemper retinopathies. This distribution and sequence of the degeneration, together with the lesions in the blood vessel walls, suggest that some vascular disturbance or blood-borne noxia may have been responsible for the retinal damage. Their nature must remain a matter for conjecture since vascular sclerosis with hypertension is uncommon in dogs; the clinical and pathological findings in this case are not, apart from the sclerosis of the choroidal and retinal blood vessels, strikingly similar to the hypertensive retinopathy produced by Keyes and Goldblatt (1937, 1938) in their experimental studies of benign and malignant hypertension in the dog.

The iridocyclitis and cataracts appeared when the retinal degeneration was advanced and are probably secondary manifestations.

Until more information is available regarding the aetiology and early development of these types of progressive atrophy, their position in any general classification of the degenerations of the dog retina must remain tentative.

Summary

(1) Three cases are described of generalized progressive atrophy of uncertain aetiology of the dog retina.

(2) Two types of the syndrome are recognized, familial and idiopathic.

(3) The familial syndrome is very similar to the hereditary atrophy but its onset occurred later in life and the disease was not reproduced by test-matings with Red Irish Setters affected with the hereditary disease.

(4) In the idiopathic type the atrophy is unsymmetrical, being least severe over the peripapillary tapetal fundus. The distribution and sequence of the retinal changes, together with the presence of degenerative changes in the walls of the retinal and choroidal blood vessels, suggest a vascular origin.

(5) Cataractous changes in the lens were present in the late stages of both types. No other symptom of ill-health was noted.

(6) Possible causes of these syndromes and their relation to other retinal degenerations in the dog are discussed briefly.

I am indebted once again to Dr. Katharine Tansley and Dr. L. C. THOMSON for their help with the histology and the electroretinography, and to Mr. C. F. Hart for the preparation of the sections.

REFERENCES

KEYES, J. E. L., and GOLDBLATT, H. (1937). *Arch. Ophthal. (Chicago)*, **17**, 1040.
——— and ——— (1938). *Ibid.*, **20**, 812.
PARRY, H. B. (1953a). *British Journal of Ophthalmology*, **37**, 385.
——— (1953b). *Ibid.*, **37**, 487.
——— (1953c). *Ibid.*, **37**, 670.
——— (1954a). *Ibid.*, **38**, 295.
——— (1954b). *Ibid.*, **38**, in the press.
———, TANSLEY, K., and THOMSON, L. C. (1953). *J. Physiol. (Lond.)*, **120**, 28.

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RADIOACTIVE PHOSPHORUS AS AN AID TO THE DIAGNOSIS OF MALIGNANT MELANOMA OF THE EYE*

BY

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THE object of this investigation was to study the usefulness of radioactive phosphorus, ^{32}P, as an aid to the diagnosis of malignant ocular melanomata. These tumours, particularly when choroidal, may present diagnostic problems; details of the tumour may be obscured by detachment of the retina, and, less commonly, the tumour may be completely hidden by opacity of the ocular media. An example of its unsuspected presence is given by Neame and Khan (1925) in a report on the pathological examination of 402 enucleated eyes, which had been blind and painful. Forty of these globes contained a malignant melanoma and in at least sixteen of the forty, no suspicion of the growth had been entertained.

Previous Investigations

A radioactive material administered to a patient is distributed throughout the body in the same way as the stable element from which it has been derived, and may subsequently be detected by external observation with a Geiger counter.

Many attempts have been made to demonstrate a difference between the uptake of radioactive material by a tumour and by normal tissue adjacent to it. Low-Beer (1946) showed that most superficial breast carcinomata have a detectable differential uptake of ^{32}P. Since then, radioactive isotopes used by other workers include copper (^{64}Cu), gallium (^{72}Ga), and iodine (^{131}I), which is usually incorporated in diiodofluorescein. Tumours investigated include those of the stomach (Gray and others, 1949), testis (Roswit and others, 1950), central nervous system (Selverstone and others, 1949), and bone (Mulry and Dudley, 1951).

Skin melanomata have been studied by Marcus and Rotblat (1950) using ^{64}Cu, ^{131}I, and ^{32}P. They found ^{32}P to be the most satisfactory material for the test; a significantly higher uptake was found in the melanoma than in the immediately surrounding skin. The concentration of phosphorus in the tumour could be resolved into two parts, the first occurring a few minutes after the intravenous injection of ^{32}P and being probably due to the increased vascularity of the tumour area. The second rise occurred after more than 3 weeks and represented a genuine uptake of ^{32}P by the tumour cells.

Ocular tumours, usually malignant melanomata, have been investigated by Trevor-Roper and others (1952), Thomas and others (1952), and Town (1952). The first of these groups used diiodofluorescein labelled with ^{131}I and found no

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significant increase of radioactivity over the tumour area as compared with a corresponding site in the other eye.

Thomas and others (1952) and Town (1952) have published preliminary reports on the use of ^{32}P, which they have found to be a valuable aid to the diagnosis of ocular tumours. In both investigations a Geiger counter was applied to the eyes one hour after the intravenous administration of ^{32}P. Thomas and others (1952) report the maximum reading, in counts per min. obtained from both eyes in each of seven patients. In one other case all the readings appear to have been taken from the affected eye. In these patients there were four cases of malignant melanoma, and one of suspected metastatic carcinoma; the remaining three had simple unilateral ocular lesions. The Geiger counter readings for the four cases of malignant melanoma were from 50 to 100 per cent. greater over the tumour than over the corresponding normal area. In the other four cases the readings over compared areas varied by up to 25 per cent.

Town (1952) gives readings taken from eight cases, of which four had malignant ocular tumours, while the other four had non-malignant unilateral ocular lesions. In the non-malignant cases the maximum difference in the readings between the two eyes was about 50 per cent., and in two of the malignant cases there was a similar variation. In the other two malignant cases the readings in the affected eye were generally 50–100 per cent. more than in the compared areas.

Material and Method

In the present investigation it was considered advisable to observe the ocular uptake of ^{32}P in persons with healthy eyes, before studies were carried out on patients with suspected malignant melanoma. Tests were made with four patients in a surgical ward (Prof. C. F. W. Illingworth, Western Infirmary, Glasgow) who were receiving ^{32}P for another investigation.

The method used was similar to that of Thomas and others (1952). A dose of 100 microcuries* of ^{32}P was injected into an arm vein and cocaine hydrochloride drops 2 per cent. was instilled into each conjunctival sac. Readings with the Geiger counter† were taken for periods of a minute from alternate eyes at anatomically similar positions and were repeated at frequent intervals during a period of 2 hours after the time of injection. Readings so obtained are shown in Table I. In each case the background counting rate was measured and found to be negligible.

The following observations are made in connection with Table I. The Geiger counter was applied directly over the eyeball and held there for as long as the observer desired, in the absence of epiphora or ocular movement of the patient. It is interesting to note that an appreciable reading was obtained over the eye within 15 seconds of the administration of ^{32}P.

In Case 4 the left pupil was dilated with a mydriatic, with no apparent influence on the counter readings; the cases of suspected malignant melanomata later examined were all atropinized before the counting tests began.

*A dose of 100 microcuries, or at the most 200 microcuries, is enough for an investigation of this kind. Both Thomas and others (1952) and Town (1952) report that they gave a dose of 500 millicuries; more probably they gave 500 microcuries.

†The Geiger counter tube was supplied by Twentieth Century Electronics, Ltd., New Addington, Surrey (type MB4H). The sensitive volume is a glass cylinder with, at one end, a very thin window having an effective diameter of about 6 mm. B-rays from ^{32}P cannot penetrate the side wall of the counter and can enter it only through the end window. The average range in tissue of the ^{32}P B-rays is about 2 mm.; the counter therefore records the amount of ^{32}P in a region under the end window, with a diameter of about 6 mm. and a depth of about 2 mm.

TABLE I

COUNTING RATES OBSERVED IN FOUR NORMAL PERSONS BY APPLICATION OF A GEIGER COUNTER TO COMPARABLE AREAS OF BOTH EYES, AFTER INTRAVENOUS INJECTION OF 100 MICROCURIES OF ^{32}P.

Figures represent counts per minute. R=right eye, L=left eye.

Time after Injection (min.)	Case 1		Case 2		Case 3				Case 4			
	Central		Central		Nasal		Temporal		Nasal		Temporal	
	R	L	R	L	R	L	R	L	R	L(1)	R	L(1)
5	119	93	86	106	139	137	173	160	250	214	184	181
15	144	153	156	156	143	197	161	172	144	200	190	142
30	124	107	194	415(2)	227	153	175	167	112	170	200	186
35	—	—	136	143	—	—	—	—	—	—	—	—
60	215(3)	88	174	155	204	175	181	199	260(4)	311(4)	202(4)	199(4)
120	92	91	137	134	268	213	201	180	195	210	229	248

(1) Left eye dilated with atropine sulphate drops, 1 per cent.
(2) Epiphora.
(3) *Epiphora*; blotting paper soaked in tear fluid at this time gave a counting rate of 60 per min.
(4) Immediately after injection of 2.5 mg. histamine acid phosphate (given for another investigation).

Epiphora causes a significant increase in the activity recorded by the counter, and the tear fluid itself, if collected on a piece of blotting paper and applied to the *Geiger counter, gives an appreciable reading.* It is, of course, to be expected that ^{32}P will appear in all body fluids shortly after administration.

The fluctuations in counting rate shown in Table I are quite substantial, but are not significant. They are due partly to the random nature of the counting process and partly to small movements of the eye and local variations in the circulation.

Tests were then made with four consecutive cases of suspected malignant melanoma of the eye, in patients admitted in 1953 to the Glasgow Eye Infirmary. The clinical details are given in Table II.

TABLE II

COUNTING RATES OVER COMPARABLE SITES IN DISEASED AND HEALTHY EYES IN FOUR CASES OF OCULAR MALIGNANT MELANOMA

Figures represent counts per minute. R=right eye, L=left eye. Left eye dilated with atropine sulphate drops (1 per cent).

Time after Injection (min.)	Case 1				Case 2				Case 3		Case 4	
	Nasal		Temporal		Nasal		Temporal		Nasal		Nasal	
	R	L (affected)	R	L	R	L	R	L (affected)	R	L (affected)	R	L (affected)
From 60 to 120	148	204	252	161	180	180	240	150(1)	78	66	210	228(1)
	169	271	151	102	270	210	330	300(1)	72	84(1)	210	240
	125	267	111	112	240	210	210	300(1)	54	60(1)	180	198
	152	260	109	108	300	300	210	210	60	60	168	258(1)
	110	230	122	84							216	240(1)
	98	93(1)										
	151	160(1)										
	164	113(1)										

(1) Directly over tumour.

Case Reports

Case 1, male, aged 54, complained of dimness of vision of the left eye and left-sided headache of 3 weeks' duration. On examination, the right eye was healthy in every respect. The patient could only perceive hand movements at half an inch with the left eye, and external examination of the left eye was normal, as was the ocular tension recorded on a Schiotz tonometer. Ophthalmoscopy revealed a solid-looking globular detachment of the retina in the lower nasal quadrant extending anteriorly as far as could be observed. On its surface were some dilated retinal blood vessels. There was old choroidal degenerative change in the upper nasal quadrant peripherally. No retinal tear was seen. Transillumination showed a shadow in the area of retinal detachment. General medical examination revealed the presence of a firm enlarged liver and of melaninuria.

Enucleation of the left eye was performed.

Pathological Report (Dr. J. D. Fraser):
The globe was cut just above the horizontal meridian. There is a necrotic choroidal tumour extending from the optic nerve forward to the equator in the lower nasal quadrant. There is detachment of the retina.

Histological Report: The appearances are those of a choroidal sarcoma. The cells are of amixed type—some being small round ones and some larger. Necrosis in the tumour is a marked feature. Extra-ocular spread through the scleral lamellae is present.

Case 2, male, aged 55, complained of flashes of light in front of the left eye of about one year's duration, and dimness of vision of the left eye of several weeks' duration. The right eye was healthy. On examination, the visual acuity of the left eye was 4/60. External examination showed a few dilated conjunctival and episcleral vessels near the limbus at " 3 o'clock ". Ocular tension (Schiötz) was normal. Ophthalmological examination showed large vitreous opacities and a fine vitreous haze—probably due to red blood cells. A globular retinal detachment was present on the temporal half of the retina, fringed by retinal haemorrhages below and peripherally. Transillumination showed a slight shadow below and no shadow temporally. Several days later a massive vitreous haemorrhage occurred obscuring all detail. Nothing abnormal was found on general medical examination.

Enucleation of the left eye was performed.

Pathological Report (Dr. J. D. Fraser):
The globe was cut in the oblique meridian. There is a mushroom-shaped tumour of the choroid in the region of the equator on the temporal side of the globe, causing a primary and secondary detachment of the retina. There is haemorrhage into the vitreous.

Histological Report: The histological appearances are those of a choroidal sarcoma in which a recent haemorrhage has occurred. In addition there is a recent sub-retinal exudate. The angle of the anterior chamber is narrowed as in glaucoma. There is no evidence of extra-ocular spread.

Case 3, male, aged 49, complained of dimness of vision of the left eye of 2 or 3 months' duration. The right eye was healthy. Vision in the left eye was reduced to hand movements in the temporal periphery. Ocular tension (Schiötz) was normal. External examination of the eye revealed no abnormality. Ophthalmoscopic examination showed a large globular retinal detachment in the upper nasal quadrant overhanging the disc and macular area, and a shallow retinal detachment nasally below. After rest in bed for 4 days the shallow retinal detachment disappeared and the large detachment above became considerably smaller, more grey, and solid looking. Transillumination showed an indefinite shadow in this area. General medical examination revealed no evidence of metastasis.

Enucleation of the left eye was carried out.

Pathological Report (Dr. J. D. Fraser): There is a mushroom-shaped growth extending from the equator to within 3 mm. of the nervehead on the upper nasal quadrant. The growth measures 12 mm. across and 13 mm. into the vitreous. The nervehead does not appear to be involved. The tumour is vascular, the detachment extends round the equator on the temporal side.

Histological Report: The appearances are those of a choroidal sarcoma which has ruptured Bruch's membrane. No extra-ocular spread is visible.

Case 4, male, aged 34, had always had a defective right eye. He complained of a blur in front of the left eye of 2–3 hours' duration. On examination, corrected visual acuity of the right eye was 6/18 and of the left eye 6/12. Ocular tension (Schiötz) was normal. Slit-lamp examination of the left eye showed a rounded, brown, pigmented mass in the substance of the iris from "9 to 12 o'clock", appearing to block the angle of the anterior chamber. On its surface were many dilated blood vessels and a small hyphaema was present below (Fig. 1). Apart from slight ectropion uveae, the rest of the iris was healthy. Transillumination of the mass showed no shadow. Ophthalmoscopy was negative. General medical examination showed no abnormal findings.

FIG. 1.—Case 4, left eye showing extent of melanoma in upper nasal quadrant of iris.

Enucleation of the left eye was carried out.

Pathological Report (Dr. J. D. Fraser):

There is a tumour of the iris—limited to the iris in the upper temporal quadrant and measuring 7×5 mm. across—which is occluding the angle of the anterior chamber (Fig. 2*a* and *b*). Apart from this the eye appears normal.

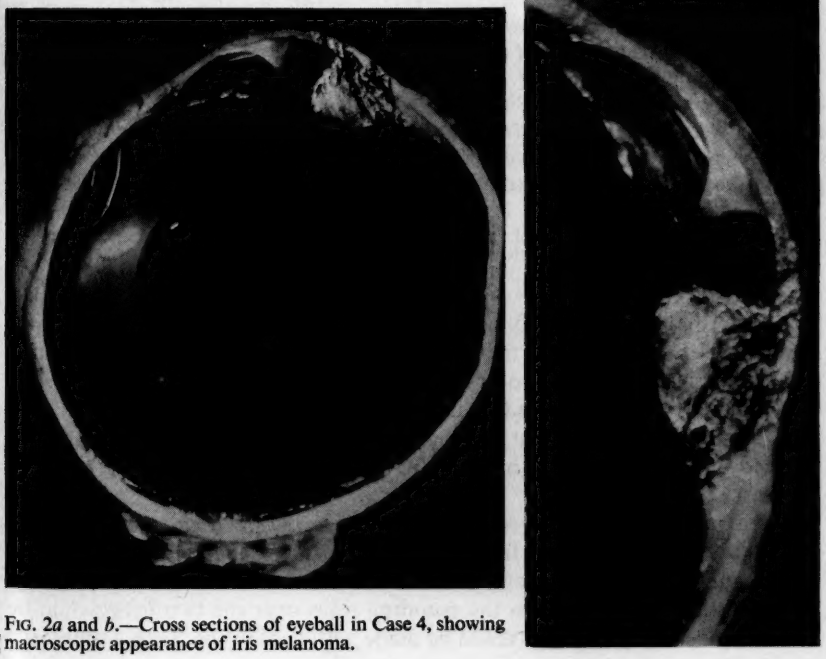

FIG. 2*a* and *b*.—Cross sections of eyeball in Case 4, showing macroscopic appearance of iris melanoma.

Histological Report: The appearances are those of a melanotic sarcoma of the iris infiltrating the ciliary body and surrounding the region of the arterial circle (Fig. 3). There is blockage of the drainage angle and infiltration of Schlemm's canal.

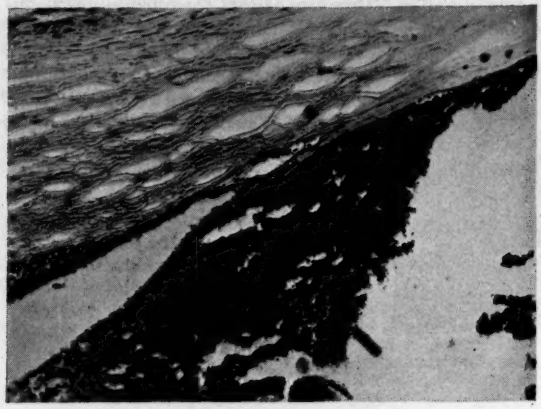

FIG. 3.—Photomicrograph of cross section through angle of anterior chamber at site of iris melanoma in Case 4 (×100).

In each of those four cases, the dose of ^{32}P was between 180 and 220 microcuries. After cocainization of the eye, the window of the Geiger counter was applied to different parts of the affected eye and to corresponding points in the other eye. The counting rates measured in this way are shown in Table II.

Only in Case 1 were the readings appreciably greater over the diseased eye. (Variations in counting rate of as much as 15 per cent. are to be expected because of the random nature of the counting process and are not significant for our present purpose). It will be observed that no enhanced activity was detected directly over the tumour itself which, in this case, was necrotic.

In Cases 2, 3, and 4, no significant difference in the counting rates was obtained, yet, in Case 4, the Geiger counter was sometimes separated from the tumour by only the thickness of the cornea. For testing the value of this method of diagnosis, no more suitable case of uveal malignant melanoma is likely to be found.

Discussion

Ocular malignant melanomata usually grow rapidly and metastases are common. Enucleation is therefore performed as soon as the diagnosis is reasonably certain. It would not be justifiable, unless the diagnosis was much in doubt, to wait for a further 3 weeks to find whether the tumour cells could metabolize significant amounts of ^{32}P, as in the skin melanomata studied by Marcus and Rotblat (1950). No such attempt has been made in the cases reported here.

Statistical analysis of the results is difficult in the presence of so many extraneous factors, including, for example, local fluctuations in the circulation, and accumulation of tear fluid on the counter window. For this reason, a very substantial difference in the counting rates over the two eyes would be necessary before a diagnosis could be made with confidence; such a difference

was not shown in the cases we have investigated—even in Case 4, where the tumour was situated only a millimetre or so from the counter window. It seems unlikely that the use of ^{32}P in this way will give useful guidance in the difficult diagnostic problems sometimes associated with choroidal tumours which most often arise in the less accessible posterior half of the globe. Nor is it known whether the use of ^{32}P will enable a distinction to be made between malignant melanomata and conditions such as acute choroiditis, where the circulation may be increased locally.

Thomas and others (1952) found the test to be useful, though some of the variations in counting rate which they considered significant are no greater than those occurring in our control series. Their use of cases of simple detachment of the retina as controls is interesting, since this condition simulates many of the effects of choroidal melanoma.

Town (1952) used a wider range of malignant tumours and as controls employed a variety of non-malignant ocular conditions. Here, too, the variations in counting rate were often no greater than in our control patients.

The negative results of Trevor-Roper and others (1952) are to be expected from consideration of the technique which they used. The Geiger counter had an aperture of diameter 2.5 cm. and the material employed (radioactive iodine, ^{131}I) gives penetrating γ-rays with a range of several cm. in tissue. The counter therefore recorded activity from a considerable mass of tissue in and near the eye, rather than from the tumour itself.

Summary

The uptake of radioactive phosphorus by the eye has been investigated in four normal patients and four consecutive cases of ocular malignant melanoma, at various times during the first 2 hours after intravenous injection. No significant difference in uptake was found between diseased and healthy eyes.

We are grateful to Dr. J. D. Fraser, pathologist to the Glasgow Eye Infirmary, for the pathological reports and the photographs, to Dr. A. Mellick, Dr. A. Wright Thomson, and Dr. J. P. White of the Glasgow Eye Infirmary for permission to use their cases, and to Professor C. F. W. Illingworth of the Western Infirmary, Glasgow, for permission to use the control cases. The counting apparatus was provided by the Physics Department of the Western Regional Hospital Board, Glasgow.

REFERENCES

GRAY, S. J., SCHULMAN, J., and FALKENHEIM, M. (1949). *Gastroenterology*, **13**, 501.
LOW-BEER, B. V. A. (1946). *Science*, **104**, 399.
MARCUS, R., and ROTBLAT, J. (1950). *Brit. J. Radiol.*, **23**, 541.
MULRY, W. C., and DUDLEY, H. C. (1951). *J. Lab. Clin. Med.*, **37**, 239.
NEAME, H., and KHAN, WAJID ALI (1925). *British Journal of Ophthalmology*, **9**, 618.
ROSWIT, B., SORRENTINO, J., and YALOW, R. (1950). *J. Urol. (Baltimore)*, **63**, 724.
SELVERSTONE, B., SOLOMON, A. K., and SWEET, W. H. (1949). *J. Amer. med. Ass.*, **140**, 277.
THOMAS, C. I., KROHMER, J. S., and STORAASLI, J. P. (1952). *Arch. Ophthal. (Chicago)*, **47**, 276.
TOWN, A. E. (1952). *W. Va Med. J.*, **48**, 356.
TREVOR-ROPER, P. D., NEWTON, K. A., and NICHOLSON, J. P. (1952). *British Journal of Ophthalmology*, **36**, 543.

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STRENGTH OF THE ORBICULARIS OCULI*

BY

H. BASIL JACOBS

London

IT was considered that some ophthalmic disorders may be correlated with variations in the strength of the orbicularis oculi. To gain some idea of the variations to be found in the strength of this muscle, a random sample of patients coming for refraction and a group of medical student volunteers were examined. Those cases were excluded which might be supposed to have some impairment of orbicularis function. It was thought that the range of findings might be not inconsiderable, that conclusions could only be drawn from relatively large numbers, and that comparison of small numbers making up sub-groups within the main body of personnel tested would not be of value.

It was found that the range of findings was even wider than anticipated; in fact so wide that the original object of the investigation to establish a norm (*viz.* a minimum and maximum between which *all* normal values would lie, values lying outside implying pathology)—obviously could not be fulfilled. Notwithstanding this negative aspect of the findings, this paper is presented to introduce a new instrument and to indicate its possible usefulness and limitations in non-ophthalmic medicine. The results are presented in outline.

Instrument

This is essentially a piston and cylinder connected to a pair of specula that are introduced into the palpebral fissure. The specula are forced together by the power of the patient's lids closing, and an air pressure is developed inside the cylinder head to oppose and overcome this (Figs 1 and 2).

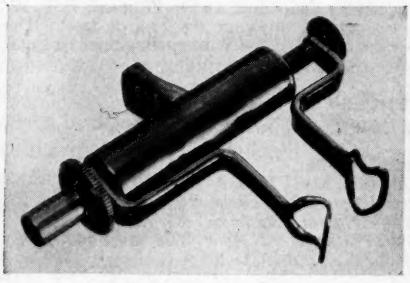

FIG. 1.—Instrument assembled for use.

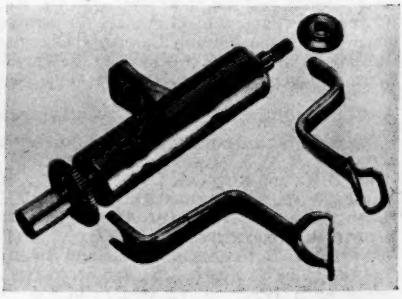

FIG. 2.—Specula detached for sterilization.

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The instrument was standardized against weights on two occasions, the second being used to check the first. Fig. 3 indicates the relation of readings to grams weight. Relevant measurements are:

Bore of cylinder	0·365 in.
Length of cylinder stroke	0·812 in.
Length of cylinder piston	0·550 in.
Size of specula	0·315×0·500 in.

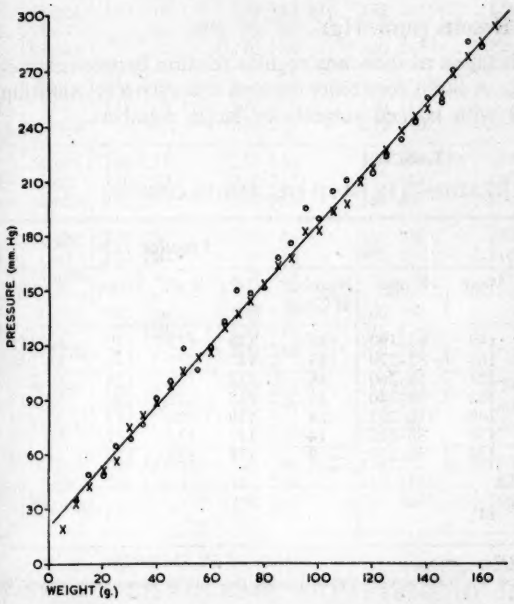

FIG. 3.—Calibration of dynamometer against weights. Dots and crosses indicate results obtained on two different occasions separated by a six months interval.

Method

The instrument is held in one hand and carefully introduced whilst the lids are manipulated with the other (Fig. 4). The subject is asked to close the eye gently, thus engaging the two specula with his lids. The cylinder is connected to a pump, an air reservoir, and a mercury manometer, The subject is then asked to squeeze his eye shut with all possible force, and the pressure in the instrument is increased until the piston just moves—*i.e.* the lids just part. The piston is tapped back with a finger and should then immediately come out again. It is at this point that the reading is taken, it being assumed that the force generated in the cylinder is

FIG. 4.—Arrangement of the apparatus for use.

equal to that exerted by the muscle. The subject must be encouraged in his efforts all the time.

Material

Group 1.—190-odd routine refraction cases seen at Moorfields Eye Hospital.
Group 2.—99 volunteer medical students.
Group 3.—7 medical students on whom serial readings were taken on different days.

Results (mm. Hg)

Group 1 (Table I).—Analysis failed to show any regular relation between refraction and the readings obtained. A slight possibility remains that such a relationship could be demonstrated either with trained subjects or larger numbers.

TABLE I

SUMMARY OF MEAN READINGS IN EACH DECADE IN GROUP 1

Decade	Males					Females				
	Number of Cases	Right *	Left †	Mean	Range	Number of Cases	Right *	Left †	Mean	Range
Second	4	158	139	149	97–190	15	140	123	132	90–115
Third	17	164	161	163	85–220	13	147	151	149	88–240
Fourth	15	122	120	121	59–260	16	134	124	129	66–210
Fifth	16	141	134	138	64–240	33	135	122	129	62–212
Sixth	9	153	145	149	110–205	24	119	106	113	47–210
Seventh	9	136	137	137	88–220	14	131	133	132	80–180
Eighth	5	138	130	134	90–210	3	138	122	130	46–190
Total...				141					120	

*Mean of right eyes = 137 †Mean of left eyes = 130

*Mean readings (compounded right and left, male and female) comparing the 3rd–7th decades are set out in Table III.

Group 2.—The range of readings obtained is indicated in Fig. 5 (overleaf), and a comparison of the males and females in Fig. 6 (overleaf).

Group 3 (Table II, opposite).—Two or three readings were taken on any given occasion. The mean and actual readings are given. In addition, readings were taken for the orbicularis oris—the method being comparable to that used for the orbicularis oculi—but the lips were pursed against the specula. Case 6 was female, the rest were males.

Inferences

(1) A Normal Value—The results in the group of medical students indicate that an average reading lies between 120–140 mm. Hg with a wide range of readings above and below; this figure is obtained in apparently normal subjects. This well brought out by Fig. 5 (overleaf).

(2) Variations

(*a*) *Age*—Only in the 3rd–7th decades are the numbers sufficient to make

TABLE II

RESULTS IN GROUP 3

Case No.		Actual Readings				Mean Readings			
		Day 1	Day 2	Day 3	Day 4	Day 1	Day 2	Day 3	Day 4
1	Right	120 116 118	138 114 116	132 104 115	126 108	*118*	*123*	*117*	*117*
	Left	94 88	80 86	105 96 82	86 98	*91*	*83*	*94*	*92*
	Mouth		170 180	240 245	236 226		*175*	*242*	*231*
2	Right	144 120 124	160 150	168 110 120		*129*	*155*	*133*	
	Left	115 124 116	128 118	130 118		*118*	*123*	*124*	
	Mouth		230 235	220 246			*232*	*233*	
3	Right	142 155	155 136	174 148 154	124 138 148	*148*	*145*	*159*	*137*
	Left	120 130	155 135	172 168	146 125 130	*125*	*145*	*170*	*117*
	Mouth	210 214	228 238	282 260	226 218	*212*	*233*	*271*	*218*
4	Right	125 126	166 156 154	156 132	130 144	*125*	*159*	*144*	*137*
	Left	124 130	138 136	142 132	116 118	*127*	*137*	*137*	*117*
	Mouth		250 215	215 230	216 220		*232*	*222*	*218*
5	Right	170 158	190 172	160 168		*164*	*181*	*164*	
	Left	128 128	130 138	136 126		*128*	*134*	*131*	
	Mouth		300 288	260 238			*294*	*249*	
6	Right	136 138	138 156	188 202	150 162	*137*	*147*	*195*	*156*
	Left	105 105	85 95	138 130	140 142	*105*	*90*	*134*	*141*
	Mouth			280 260	226 204			*270*	*215*
7	Right	166 135 146	110 96 112	124 132	118 125	*149*	*105*	*128*	*121*
	Left	118 132 138	104 102	112 118	82 94	*129*	*103*	*115*	*88*
	Mouth		198 228	284 288	268 278		*213*	*286*	*273*

TABLE III

VARIATION OF READINGS WITH AGE OF PATIENTS

Decade	No. of Cases	Mean Readings
3rd	30	157
4th	31	125
5th	49	132
6th	33	122
7th	23	134

tentative comparisons. The figures in Table III do not reveal the expected variation with age. The high average in the 3rd decade (compared with 124 in the 99 medical students) makes one suspect some undetermined heterogeneous factor.

(*b*) *Sex.*—Both Groups 1 and 2 show that the orbicularis oculi tends to be stronger in males than females.

(*c*) *Side.*—There is no consistent preponderance of right over left among the medical students. The figures in the other groups are not considered reliable in this respect as the right side was always done first and a fatigue factor may have entered into the second reading on the left side. On the other hand, among the students, in cases where there was a marked difference between the two sides, the results were generally checked after a few minutes—the machine being pumped up to about two-thirds of the expected reading and the left side done first, so tending to reduce the fatigue factor.

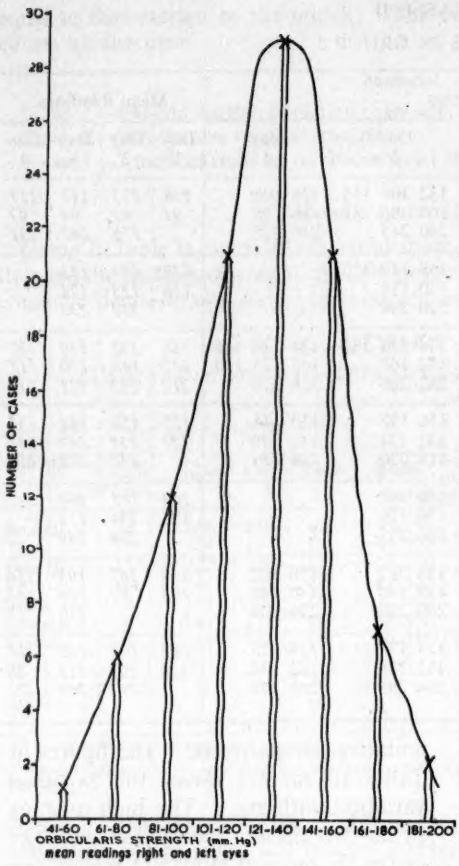

FIG. 5.—Mean strength of orbicularis oculi in 99 students (60 male and 39 female).

(*d*) *Period.*—Serial readings showed that readings obtained on different days may be consistent or variable, depending on the subject. Indeed, some variation—generally not great—may be shown in different readings on the same day. Of the seven students, two gave fairly consistent readings (1 and 5), three gave readings that were broadly of the same order (2, 3, and 4), and two gave irregular readings (6 and 7). One of the most irregular (6) was the only female.

Comment

(1) It must be emphasized that the normal values noted above are based on results obtained from inexperienced subjects at a first testing. Possibly—in fact, very probably—a different average would be obtained with trained subjects.

(2) Müller and Droescher (1938) gave the strengths of the orbicularis oculi obtained by using a dynamometer working on a spring principle. An average reading of 160 G (this series 60–70 G) with a range of 134–209 G (this series 15–145 G) was found. The greatest difference between the two sides was 25 G (this series, 30 G—in a man who admitted slight epiphora on the weaker side and also had chronic antral trouble on that side). Springs that measure large ranges of small forces are unreliable, and the figures obtained with an instrument working on this principle probably reflect this fact. Further, a sudden thrust is measured with a spring-type instrument, whereas the air dynamometer requires a thrust which is sustained to some degree. Notwithstanding this, the air-type instrument can be used (by previously mounting the pressure) to measure a thrust. The difference (generally a higher reading) does not compare with the difference between Müller and Droescher's series of figures and our own. Also the exact size of the spring instrument—in

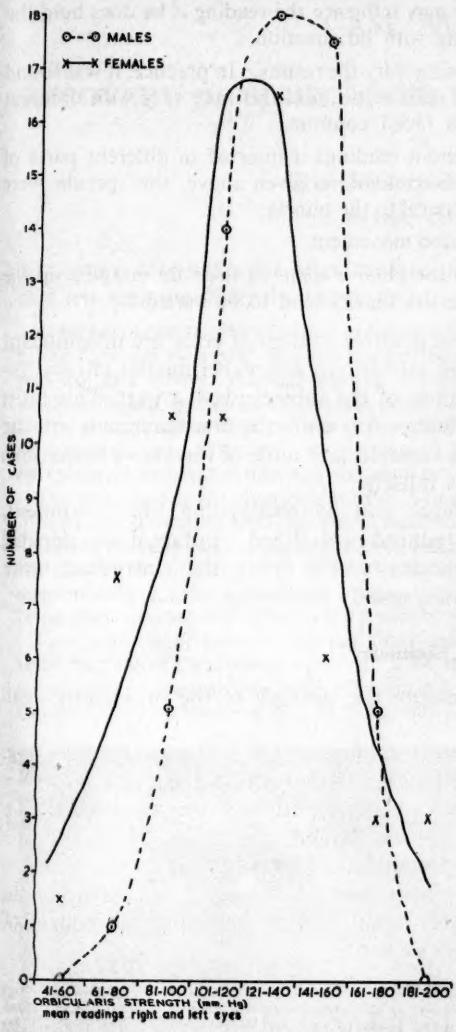

FIG. 6.—Mean strength of orbicularis oculi in 60 males and 39 females. The number of females has been scaled up 50 per cent. for sake of comparison.

particular of the specula—is not described.

(3) Although it had been anticipated at the inception of this investigation that a wide range of not necessarily repeatable results would be obtained (the subject's volitional powers being on test as well as his muscle power), the extreme range of variation actually obtained (50–186 mm. Hg) was a surprise. It meant that no lower limit of normal could be proposed, and that normals and known abnormals tended to overlap in this particular determination. Therefore, it was decided to curtail the much wider investigation to establish a norm as originally envisaged. The foregoing implies that any single determination in an individual can have but limited significance; but, although this is true, it is also true that the comparison of bulk determinations in given clinical groups may yield interesting data.

(4) The data obtained in a given individual, provided each reading is confirmed by taking an average of two or three determinations, may be revealing by estimating the response of a given patient to such drugs as prostigmine and muscle relaxants.

Limitations of the Method

(1) A defect of the instrument is that the point at which the piston "fires off" varies with the rate of rise of pressure in the cylinder. In using it the operator always made an effort to make the rate of increase of pressure fairly even.

(2) It is possible that the operator may influence the reading if he does hold the instrument so as to avoid interfering with lid function.

(3) Tilting the instrument may possibly vary the results. In practice, it was found that the instrument tends to sit at a definite tilt, and this may vary with different patients—presumably depending on facial contour.

(4) The instrument may give different readings if inserted in different parts of the palpebral fissure. In all the determinations given above, the specula were engaged at the medial canthus just lateral to the puncta.

(5) There is a slight shear in the piston movement.

(6) Some subjects tend to extend the *point d'appui* of their lid margins up the shank of the instrument as in them the shanks tend to be buried.

It is considered that all the above possible sources of error are insignificant compared with the errors that are intrinsic in any experimental clinical investigation in man where the volition of the subject plays a part. This must be the major source of unreliability—for even the measurements of the strength of the orbicularis oris are variable, and most of the above limitations do not apply in dealing with this muscle.

By slight variations in technique and by redesigning the instrument, factors 1, 2, 3, 4, and 5 could be reduced or nullified. In fact it was decided to stick to one technique and one instrument rather than introduce more variables and make the investigation unduly prolonged.

Summary

An instrument designed to measure the strength of the orbicularis oculi is described.

In 99 medical students, an average finding of 120–140 mm. Hg (60–70 g. weight) was obtained with a wide range on either side of the mean.

A sex variation was found—males being slightly stronger than females.

No consistent variation with age was found.

No general right- or left-sided preponderance was found.

It is considered that the instrument may prove useful in estimating the effects of drugs on the orbicularis oculi, and in following the course of recovery in cases of such disorders as Bell's palsy.

I am grateful to Sir Stewart Duke-Elder and Dr. E. C. Thomson of the Institute of Ophthalmology for advice and encouragement. In addition, my thanks are due to the staff of Moorfields Hospital, and to Mr. A. S. Philps, Dr. Coulson, and the Dean of St. Bartholomew's Hospital, London, for extending to me the facilities to undertake this work. I should also thank Miss Banks for her help.

REFERENCES

MULLER, H. K., and DROESCHER, J. (1938). *Ber. ophthal. Ges. Heidelberg*, **52**, 438.

Brit. J. Ophthal. (1954) **38**, 567.

CASE NOTES

RECOVERY OF SIGHT AFTER RUPTURE OF THE GLOBE*

BY

CHRISTOPHER HEATH

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RECOVERY of good sight after rupture of the globe with expulsion of lens and iris seems sufficiently unexpected to be of interest.

A farmer, aged 44, was admitted to hospital on September 2, 1952, after being kicked in the right eye by a cow. The left eye had been lost 10 years before from injury by a pick-axe in a quarry. The conjunctiva was seen to be lacerated above and the globe partly collapsed, but no further details could be made out on preliminary examination. Under general anaesthesia the wound was cleared of debris and blood-clot, revealing a rupture of the sclera in the ciliary region from 11.30 to 2.30 o'clock. The wound was closed by episcleral mattress suture and the conjunctiva repaired.

The dressing was not changed until the third day, when the globe was found to be reformed, and the cornea clear, but only a uniform obscurity of blood within.

During the next week a clearing showed behind the cornea and gradually enlarged as though the blood were being pressed uniformly backward, limiting the clear anterior segment with an even background. A fortnight after injury, the front half of the globe being clear, the rear impenetrably filled with blood, the patient was sent home.

Four weeks after injury a dull red reflex could be seen. A month later the fundus could be glimpsed and vision with +10·5 D. sph was just 6/60. Five months later the rather murky vitreous, with large white floaters, allowed a fair view of apparently undamaged fundus; vision with +11·5 D. sph. $\bar{c}$ +3 D. cyl. axis 17° was 6/9 and with +4 addition J.1 with difficulty.

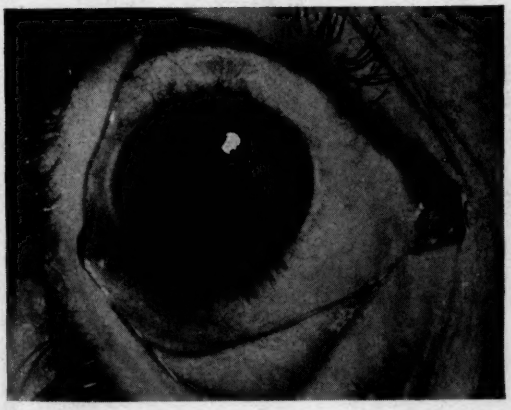

FIGURE.

At the last examination in December, 1953, 15 months after the injury, corrected vision was 6/6 partly and J.1 with ease. The eye looked curiously featureless, the only interruptions in the media being where the clear anterior segment met the smooth face of the vitreous and a few thin, papery fronds, apparently anchored in the ciliary region. The vitreous, now clear, appeared less dense than usual, the fibrillae sparser and more tenuous. The fundus, clearly seen, showed little evidence of damage; in the extreme periphery was one small white mound and in one or two areas a trace of faint pigmentary disturbance.

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PRIMARY TUBERCULOSIS OF THE CONJUNCTIVA TREATED WITH STREPTOMYCIN AND P.A.S.*

BY

STELLA EADIE

Edinburgh

PRIMARY tuberculosis of the conjunctiva is a rare condition, the existence of which was for long open to question. It has, however, now been shown that the disease can be produced experimentally (Igersheimer, 1922) and later reports have established its clinical occurrence. (Blevgad, 1933; Bruce and Locatcher-Khorazo, 1947).

Since the advent of antibiotics, there have been several reports of cases treated with streptomycin. In some, both local and systemic streptomycin has been used (Das Gupta and Usman, 1949; Sykowski, 1950); and in others the use of the drug has been combined with ultra-violet light therapy (Kamel, 1950), or with para-amino-salicylic acid (Wheeler and Sinclair, 1952). In all cases, however, the course of treatment has been comparatively short, and it has not always been effective in preventing caseation of the regional glands (Donegan, 1950).

Case Report

A girl, aged 15, was first seen at the Royal Infirmary, Edinburgh, on March 19, 1953.

History.—She had had a right cervical adenitis 3 weeks before the examination. After 10 days this had partially subsided, but the right conjunctiva was hyperaemic and 2 days later a swelling was seen under the right upper lid. Since then the condition had remained unchanged and the patient had felt well.

FIG. 1.—Inflammatory mass in upper fornix of right eye.

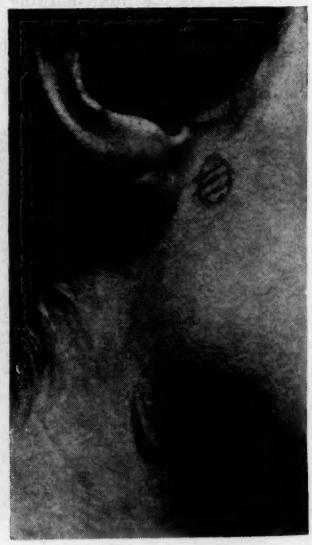

FIG. 2.—Palpable gland outlined on skin.

*Received for publication March 8, 1954.

Past History.—Nothing of importance.

Family History.—One brother suffered from pulmonary tuberculosis and was being nursed at home.

Examination.—The patient was a girl of average build, who appeared in good health.

Right eye.—The conjunctiva showed a follicular reaction above the tarsal plate and in the upper fornix. There was an irregular inflammatory mass extending from the limbus to the fornix, mainly on the medial side. This mass was fixed to the sclera and the surface showed small ulcerated areas (Fig. 1). The right pre-auricular and upper cervical glands were enlarged but not tender (Fig. 2). Visual acuity in the right eye was 6/5, and J 1 unaided. Fundus normal.

Left eye.—There was no abnormality and visual acuity was 6/5 and J 1 unaided. The fundus was normal.

She was admitted to hospital on March 21, 1953. Throughout her stay she was afebrile.

Special Investigations

Mantoux Test: 1: 10,000—strongly positive.

X ray of Chest: Negative.

Tissue Biopsy from Right Conjunctiva:

Pathological Report.—The tissue contains several typical tuberculous follicles. These are discrete and fairly recent with commencing central caseation in some of them. At one point several tubercles have breeched the lining epithelium and are discharging on the surface. The tubercles are accompanied by oedema and much non-specific lymphocyte and plasma-cell infiltration (Fig. 3).

Bacteriological Report.—A scanty growth of *M. tuberculosis* (human strain) was obtained on culture.

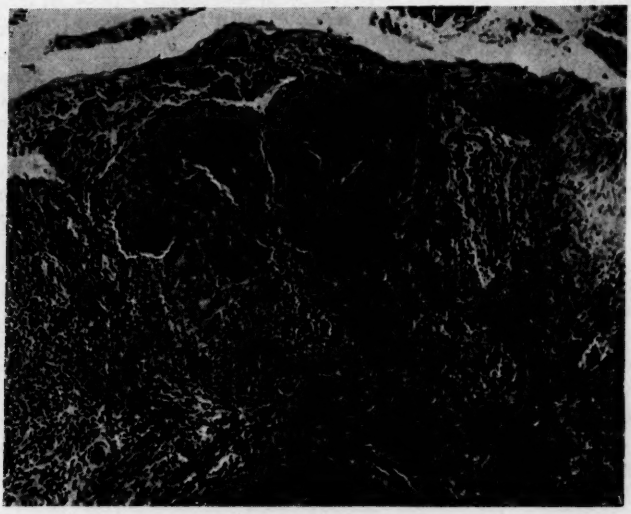

FIG. 3.—Section of tissue taken for biopsy showing tuberculous follicle formation.

Treatment.—She was given streptomycin 0·75 g. per day, and P.A.S. 15 g. per day.

On April 24, 1953, she was transferred to the Astley Ainslie Hospital where she was treated under a sanatorium régime.

In August, 1953, the streptomycin was reduced to 0·75 g. twice weekly, and in September she was discharged from hospital.

Streptomycin and P.A.S. were stopped in December, 1953.

Progress.—May, 1953, the inflammatory mass was still present but was smaller and less congested. The pre-auricular and cervical glands were smaller.

July, 1953, the conjunctival mass was resolving; the pre-auricular gland was just palpable, and the cervical gland was smaller.

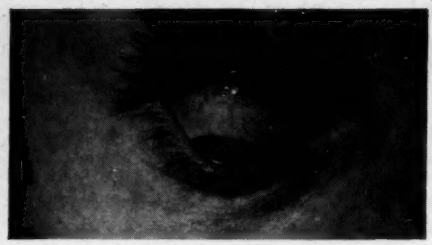

FIG. 4.—Right eye in December, 1953, showing complete resolution of inflammatory mass.

September, 1953, the conjunctiva was completely healed.

December, 1953, the eye appeared normal apart from a small scar where the biopsy had been taken (Fig. 4). A very small cervical gland could still be palpated.

Summary

A case of primary tuberculosis of the conjunctiva successfully treated by systemic streptomycin and P.A.S.

I wish to thank Dr. G. I. Scott of the Eye Department, Royal Infirmary, Edinburgh, and Professor John Crofton of the Department of Tuberculosis, University of Edinburgh, for permission to publish this case.

REFERENCES

BLEGVAD, O. (1933). *Acta ophthal. (Kbh.)*., **11**, 345.
BRUCE, G. M., and LOCATCHER-KHORAZO, D. (1947). *Arch. Ophthal. (Chicago)*, **37**, 375.
DAS GUPTA, B. K., and USMAN, M. (1949). *British Journal of Ophthalmology*, **33**, 501.
DONEGAN, J. M. (1950). *Amer. J. Ophthal.*, **33**, 1117.
IGERSHEIMER, J. (1922). *Klin. Mbl. Augenheilk.*, **69**, 226.
KAMEL, S. (1950). *British Journal of Ophthalmology*, **34**, 322.
SYKOWSKI, P. (1950). *Amer. J. Ophthal.*, **33**, 1291.
WHEELER, J. R., and SINCLAIR, S. R. (1952). *Trans. ophthal. Soc. U.K.*, **72**, 3.

Brit. J. Ophthal. (1954) **38**, 571.

CHOROIDAL DETACHMENT FOLLOWING OPERATION FOR DETACHED RETINA*

BY

J. GIBSON MOORE

London

THE incidence of choroidal detachment following operation for detached retina is not easy to assess and reports in the literature are very scanty. It probably occurs quite often where free drainage of the sub-retinal fluid is made and the eye is in a state of hypotension, but passes unnoticed unless an examination is made immediately.

Case Report

A married female aged 51 noticed flickering spots in front of the right eye, succeeded by a shadow in the lower and outer field of vision.

She was first examined on March 21, 1953, 5 weeks after the onset and a shallow retinal detachment was noted in the upper nasal quadrant. She was emmetropic with 6/6 vision in each eye. Repeated examination revealed no hole and a period of rest in bed did not influence the appearance.

At operation the area of the detachment was covered with diathermy punctures spaced at intervals of about 3 mm. extending back 14 mm. in the shape of a semi-circle from the ora serrata. There was quite free drainage of sub-retinal fluid and the retina appeared flat. Air was not injected into the vitreous and the eye was soft at the end of the operation.

The convalescence was uneventful, but, when detailed examination was made 7 days later, two small choroidal detachments were noted, one in the supernasal quadrant and the other in the supertemporal quadrant, separated by a definite interval. It was not likely that these were due to actual hæmorrhage at the time of operation as the supernasal one was not over the site of the detachment.

The anterior chamber remained of normal depth the whole time and it was not until 8 weeks after that the retina was seen to be in position where it has remained ever since.

Discussion

The unusual nature of this occurrence leads one to consider how it could develop. In the many papers dealing with choroidal detachment following glaucoma and cataract operations the most commonly quoted theories are:

(1) Formation of a cyst in the retina (Haab, 1891).

(2) Cyst-like detachment in the epithelium of the ciliary body (Velhagen, 1897).

(3) Serous exudate from the choroidal blood-vessels under conditions of abnormally low tension (Hudson, 1914; O'Brien, 1935).

(4) Formation of a rent in the angle of the anterior chamber and the seepage of fluid into the perichoroidal space (Fuchs, 1900; Rycroft, 1943).

Taking the last theory first, it is attractive to have a definite pathology, *i.e.*, a rent which can be visualized, but there are several objections:

(*a*) If a rent is present the aqueous must now pass in two directions, both through the external wound made at the limbus (in glaucoma and cataract operations) and also backwards into the perichoroidal space, and this is difficult to understand.

(*b*) The rent must be made at the moment when the anterior chamber is lost at

*Received for publication March 1, 1954.

operation. When a very hard eye is opened it is very noticeable that no tremendous mechanical stresses of an explosive nature are set up, such as one might expect and would be required by this pathology. Instead the aqueous flows out quite quietly while the anterior chamber is lost.

(*c*) Only one case of choroidal detachment has been noted after cyclodialysis (Heine, 1905) in many hundreds of cases. Rycroft (1943) has considered the rent theory favourably and in detail but with an open mind records this objection.

No-one has ever seen this rent and its existence is hypothetical. This does not, of course, exclude its possibility, but it is necessary in any valid hypothesis for the physical facts to accord with the theory. If one tries to visualize what happens when the anterior chamber is lost during a glaucoma or cataract operation it is true that the lens moves anteriorly and with it the iris. The iris is by no means a flimsy membrane and it is well accustomed to variations in position. It would require to be in strong miosis and firmly fixed at the pupillary margin for there to be any strain such as might produce a rent at its root. It is unfortunate that it is, of course, impossible to examine the angle when the anterior chamber is absent as this would clinch the matter, and even when no rent is found when the chamber is examined gonioscopically after reformation it is possible that it is then not visible through the anterior synechia which commonly form peripherally after operation.

The third theory merely requires a condition of prolonged hypotension; the choroidal detachment being produced by serous exudate from the choroidal vessels and it is the only reasonable theory that could explain its occurrence after operation for retinal detachment. O'Brien (1935) states that the fluid aspirated is derived from blood and not from aqueous, and this, of course, is very definite evidence that the fluid is derived from the choroidal veins. Duke-Elder (1940) writes:

> Owing to the sudden lowering of the intra-ocular pressure and the abolition of the anterior chamber allowing the lens and vitreous to move forwards, there is a transudation of fluid from the choroidal veins into the perichoroidal space.

This is the most likely explanation of the mechanism of choroidal detachment, but I feel that the forward movement of the lens and vitreous is not essential although it occurs in the great majority of cases because the external wound is in front of the plane of the iris. If, however, the external wound is behind the iris as in the case here described, the anterior chamber would not be lost and might even become deeper. All that is required is a condition to be set up in which the intra-ocular fluid is being lost externally at a higher rate than it can be produced by its normal sites of origin.

Summary

A case of choroidal detachment following operation for retinal detachment is reported. It is considered that this occurrence tends to disprove the rent theory in the formation of choroidal detachment.

REFERENCES

DUKE-ELDER, S. (1940). " Text-book of Ophthalmology ", vol. 3, p. 2540. Kimpton, London.
FUCHS, E. (1900). *v. Graefes Arch. Ophthal.*, **51**, 199.
HAAB, O. (1891). *Beitr. Augenheilk.*, **1**, heft 2, p. 23.
HEINE, L. (1905). *Ber. ophthal. Ges. Heidelberg*, **32**, 3.
HUDSON, A. C. (1914). *Roy. Lond. ophthal. Hosp. Rep.*, **19**, 301.
O'BRIEN, C. S. (1935). *Arch. Ophthal. (Chicago)*, **14**, 527.
RYCROFT, B. W. (1943). *British Journal of Ophthalmology*, **27**, 283.
VELHAGEN (1897). *Zbl. prakt. Augenheilk.*, **21**, 363.

BOOK REVIEWS

Text Book of Ophthalmology, Volume VI. Injuries. By Sir Stewart Duke-Elder. 1954. Pp. 1198 +xxviii, 1145 illus. (144 col.). Kimpton, London. (105*s*.).

To describe this volume " with honour due " would require something that the reviewer lacks. It would need

> An eloquence scarce given to mortals, new,
> And undebased by praise of meaner things,

because Sir Stewart's new work enormously adds to the debt which ophthalmologists owe him, whatever may be their native language. Before he has read many pages, every reader will surely feel a surge of gratitude at being supplied with such a wealth of cumulative experience, and so rich an array of pictures. Sir Stewart has generously acknowledged the help given by his colleagues at the Institute of Ophthalmology in London University, and Dr. Norman Ashton and Dr. Peter Hansell deserve high praise for the illustrations prepared in their departments. Other splendid pictures have come from different countries, especially the United States; and all these adjuncts have been utilised with first-rate technical skill. The task of preparing the bibliographies alone must have been stupendous, and the outcome is worthy of congratulation. Moreover, the publisher's reader and proof-readers have acted as the finest of combs, because errors and misprints are remarkably scarce. The firm of Henry Kimpton has good reason to feel proud of this volume.

The reader's gratitude will therefore be extended to a number of other people besides Sir Stewart, and as he reads on, his gratitude will grow; but before long that emotion will be intermingled with wonder at the range of Sir Stewart's exposition. Loyally and efficiently though the author has been served by his helpers, the fact remains that he did all the writing. If we survey the literature of other branches of medicine, we shall find that the great comprehensive systems are written by teams of authors. Hence arise inequality of performance, lack of correlation, and other disadvantages which can admittedly be reduced by careful editorship; but no editor, however devoted, can rival the work of a single genius. As well expect committees to compose a Churchillian oration. In various ways, subtly and unobtrusively, Sir Stewart keeps reminding us about related subjects in other parts of this volume and of the preceding five, so that the reader's path is smoothed not only by the uniformity of scope and method, but also by the book's close-knit texture. To use another metaphor we may liken each successive section to a pebble from which, if it drops into a lake, ripples will spread fore and aft. Nearly every paragraph is evocative, and the author does not let us forget basic pathological principles, and those important vascular changes which are alike evident after physical and chemical injuries. Much of Sir Stewart's philosophy is summed up in the paragraph connecting p. 5762 with p. 5763.

In a work of such consistently high level it may seem unnecessary to select separate items, but special mention should be made of Chapter LXIV (pp. 6125–6325) which so authoritatively deals with intra-ocular foreign bodies. Here we find admirable guidance on treatment, and wise counsel about prognosis, as well as a particularly fine description of chalcosis. Chapter LXXI (pp. 6581–6861) can without exaggeration be called a masterpiece, because here the author conveys a mass of varied information without once losing his grip on our attention. One astonishing feature is the up-to-dateness of the material. The labour of preparing so large a book often precludes the insertion of recent items, but Sir Stewart and his publisher have managed to deal with papers appearing in 1953 and even 1954, as exemplified by pp. 6852–4, in which he describes the role of sanguinarine in epidemic dropsy complicated by glaucoma.

Let it not be assumed that the paragraphs in small type should be skipped. Some of the rare diseases therein described are of particular interest, *e.g.*, galactosaemia on p. 6813. Sir Stewart's sense of proportion prevents him from lingering too long in the bypaths of knowledge, but he is well aware that the citation of curiosities can serve to amuse his readers, and speed them on to more substantial fare. We have all heard of "black-out", but "red-out" (p. 6883) will be news to some of us. So will tobacco amblyopia afflicting Australian horses who have nibbled the plant *Nicotiana suaveolea* (p. 6838). Cervical migraine (p. 6368) will be unfamiliar to many and *maladie de la canne de Provence* (p. 6748) to most readers. How many of us knew that a jet of water from a fire hose could neatly enucleate an eyeball (p. 5943), or that the lacrimal gland might prolapse after a collision between two girls playing basket-ball (p. 5959). The reviewer was unaware that rodent ulcer of the cornea had been reported as a sequel of burning by chloroform (p. 6611), and that cataract extraction had provoked poliosis of the brows and lashes (p. 6354).

Some will be disquieted to hear that the variety of objects left inside an eye during operation (p. 6153) almost rivals that of intra-ocular foreign bodies entering by accident (p. 6160–1). Another alarming piece of information is that lead poisoning can be inflicted by a hat (p. 6775). Refraction changes in diabetes have been recognized for a long time, and were indeed the subject of Sir Stewart's first contribution to this journal nearly 30 years ago, but he now reminds us that transient myopia may arise from the administration of arsenicals (p. 6783) or sulphonamides (p. 6834).

Volume VI is unreservedly recommended, not only as a work of reference dealing with ocular injuries, but also as a source of joy to all who love ophthalmology. It is difficult to see how any one could improve upon the author's description of radiational cataract (pp. 6557–73) or the evolution of retinal holes (p. 5846). Nor can one imagine that anybody else could have planned the chapters with such masterly judgment. All things great and small, from the fearful hazards of atomic warfare (pp. 6575–9) to the perils of glamour-cosmetics (p. 6692), come under Sir Stewart's gaze.

> No man hath walked along our roads with step
> So active, so inquiring eye, or tongue
> So varied in discourse.

The series of affectionate tributes to his former teachers make charming preludes to Sir Stewart's new chapters. Similar protraits have appeared in the earlier volumes, and it is significant that, with all the achievements that stand to his credit, he can still accord such generous praise to other people's work. How good it is to know that Volume VI will not after all be the last of this stately series. Half-a-dozen may be a good round number, but seven is more satisfying, not merely on account of its mystical significance, but because it conjures up those *other* Seven Wonders of the World.

Anatomy of the Eye and Orbit. By Eugene Wolff. 4th. ed., 1954. Pp. viii + 492, 406 illus. (inc. 52 col.). Lewis, London. (63*s.*).

The review of a posthumous book is often difficult: *de mortuis nil nisi bonum.* But when the book is an excellent exposition of a subject wherein the author was an acknowledged master, the task is easy. Wolff's name will long be remembered for his work on the anatomy of the eye, and this text-book, which first appeared in 1933 and has now reached its fourth edition, incorporates much of his own researches and his personal assessment of the work of others. The book is already generally recognized as a classic and in the present edition has been considerably enlarged and improved. A large amount of new matter has been added, particularly concerning the anatomy of Schlemm's canal and the limbus, the minute structure of the skin of the lids, the lower visual pathways, the blood supply of the ocular motor nerves, and gonioscopic appearances at the angle of the anterior chamber. Moreover, eighty new illustrations have been incorporated, many of them derived from the Institute of Ophthalmology. The book is without question the best modern treatise on the anatomy of the eye and orbit; it is indispensable for the student; it is beautifully produced and profusely illustrated; it is a living demonstration that man's work and ideas are more lasting than man himself. It is a tragedy that the author's untimely death means that this edition will be the last that will come from his pen.

Augenhöhlenplastik. By Istvan Csapody. 1953. Pp. 102, 71 figs, bibl. Akadémiai Kiado, Budapest.

This mongraph describes the author's method in the plastic repair of contracted sockets, with a brief review of the history of such operations.

The method consists in inserting a whole-thickness skin graft sutured on to an ingenious metal speculum into the prepared socket. The procedure is clearly described and well illustrated, and is said to give a high percentage of successful results.

The operation is not technically difficult and in view of the liability of stent or plastic moulds to be extruded, the instrument described would seem to worthy of a trial.

CORRESPONDENCE

EXTRA-CONJUNCTIVAL IMPLANTS

To the Editorial Committee of the BRITISH JOURNAL OF OPHTHALMOLOGY

SIRS—The technique described by Nairac (1954) is ingenious, but founded upon a doubtful principle. Most authorities on orbital implants (movable ocular prostheses) agree that, to stand a chance of being retained for more than 5 years, an implant must be completely enclosed in both Tenon's capsule and the conjunctiva (Choyce, 1954; Roper-Hall, 1954). The union described by Nairac between tantalum and naked muscle, not covered by Tenon's capsule and conjunctiva, is very likely to fail after 1 to 2 years at the most.

The author makes no reference to numbers of cases or to the length of time which has elapsed since operation. If he is able to indicate that a substantial proportion of his implants are still *in situ* 5 years after the operation, I shall be the first to congratulate him, but I do not believe he will find himself able to demonstrate such a degree of success.

In my opinion, by adopting this technique, he has rendered his patients a disservice, because the opportunity has been missed of inserting a completely buried implant at the time of enucleation.

Yours faithfully,

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July 16, 1954.

REFERENCES

CHOYCE, D. P. (1952). *British Journal of Ophthalmology*, **36**, 123.
NAIRAC, M. L. (1954). *Ibid.*, **38**, 378.
ROPER-HALL, M. J. (1954). *Trans. ophthal. Soc. U.K.*, **74**. (In the press).

NOTES

BRITISH ORTHOPTIC JOURNAL

THE latest issue of the *British Orthoptic Journal*, which appeared in June this year, is well-arranged and illustrated, and betrays evidence of the sub-editor's work in pruning and styling. The science of orthoptics is seen to be making progress in Switzerland, Belgium, Holland, and Australia, as well as in this country, and it is plain that the orthoptists interest themselves in a wide range of allied subjects outside their specialized clinics. Since 1939, when it first came out, this journal has been limited to one number each year, but the newly-constituted editorial committee under Miss Joyce Mein's leadership hopes to produce it in future at six-monthly intervals. Despite rising costs in recent years, the annual subscription has remained at ten shillings, and the *British Orthoptic Journal* committee hopes that the policy of supplying two issues a year will not entail any increase in subscription. This hope may perhaps be translated into certainty if the list of subscribers can be augmented. Nearly all practising orthoptists already subscribe, but hitherto the support from ophthalmologists has been disappointingly small. Medically-qualified people have often supposed that the *Orthoptic Journal* consists entirely of papers dealing with technical aspects of orthoptic work; and admittedly it is impossible for most ophthalmologists to become familiar with all the routine methods of investigation and treatment

used by orthoptists. Let it be emphasized, however, that many of the papers in each issue are written by ophthalmologists, and that the subjects have included operative techniques, neuro-ophthalmology, and other matters vitally affecting an ophthalmic surgeon's work, as well as accounts of educational work with cerebral palsied and partially-sighted children. In the early days of orthoptics much effort was expended in treating certain types of squint which have since been recognized as unsuitable, but nowadays we have at our disposal a harvest of cumulative experience, so that the selection of cases has been much facilitated. Moreover the value of orthoptic investigation has been established beyond controversy. Bearing these considerations in mind, ophthalmologists should seriously consider the wisdom of subscribing ten shillings a year to the *British Orthoptic Journal*. Subscription may be sent to Miss Joyce Mein, Orthoptic Department, Royal Infirmary, Sheffield 6; she will be pleased to supply a Banker's Order to simplify subsequent payments.

Index Ophthalmologicus

A new *Index Ophthalmologicus* will be published by the International Council of Ophthalmology containing the names and addresses of ophthalmologists, and particulars of the ophthalmic societies, and the main eye hospitals and ophthalmic periodicals throughout the world. This volume will be on sale at the XVII International Congress of Ophthalmology (Montreal-New York) in September, 1954, price 2.50 dollars, or may be obtained from Dr. A. C. Copper, Nieuwstad 97, Zutphen, Holland, after the date of the congress, price 2.75 dollars.

Oxford Ophthalmological Congress

The next meeting will take place on June 28 and 30, 1955. Mr. J. W. Tudor Thomas of Cardiff has accepted the Doyne Lectureship for 1955.

Faculty of Ophthalmologists

At a council meeting held on July 23, 1954, the following officers were elected for 1954-1955 :

President: -	Mr. J. H. Doggart
Vice-Presidents :	Dr. John Marshall, Mr. T. Keith Lyle
Hon. Treasurer :	Mr. A. McKie Reid
Hon Secretary :	Mr. E. F. King

American Ophthalmological Society

Officers, 1954–55

President : - -	Everett L. Goar (*Houston, Texas*)
Vice-President : -	Alan C. Woods (*Baltimore, Md*)
Secretary-Treasurer :	Maynard C. Wheeler (*New York*)
Editor : - -	Gordon M. Bruce (*New York*)

HONOURS

The honorary degree of LL.D. was conferred upon Mr. J. W. Tudor Thomas by the University of Glasgow, on July 8, 1954.